DISSERTATION ON

"CLINICAL STUDY AND MANAGEMENT OF OBSTRUCTIVE JAUNDICE"

Submitted in partial fulfilment for the degree of

M.S.GENERAL SURGERY

BRANCH – I

INSTITUTE OF GENERAL SURGERY,

MADRAS MEDICAL COLLEGE,

THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY,

CHENNAI,

APRIL 2015

CERTIFICATE

This is to certify that the dissertation entitled "CLINICAL STUDY AND MANAGEMENT OF OBSTRUCTIVE JAUNDICE" is a bonafide original work of Dr. BALAJI.K.M, in partial fulfilment of the requirements for M.S. Branch– I (General Surgery) Examination of the Tamil Nadu Dr. M.G.R. Medical University to be held in APRIL 2015 under my guidance and supervision in 2013-14

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DECLARATION

I hereby solemnly declare that the dissertation titled "CLINICAL STUDY AND MANAGEMENT OF OBSTRUCTIVE JAUNDICE" is done by me at Madras Medical College & Rajiv Gandhi Govt. General Hospital, Chennai during 2013-14 under the guidance and supervision of Prof.Dr.P.RAGUMANI.M.S. The dissertation is submitted to The Tamilnadu Dr.M.G.R. Medical University, Chennai towards the partial fulfilment of requirements for the award of M.S. Degree (Branch-I) in General Surgery.

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CONTENTS

INTRODUCTION

Jaundice is a frequent manifestation of biliary tract disorders and the evaluation and management of obstructive jaundice is a common problem faced by the general surgeon.

Obstructive jaundice is strictly defined as a condition occurring due to a block in the pathway between the site of conjugation of bile in liver cells and the entry of bile into the duodenum through the ampulla. The block may be intrahepatic or extra hepatic in the bile duct.1.

Despite the technical advances, the operative modes of management of obstructive jaundice were associated with very high morbidity and mortality. Yet, during the last decade significant advances have been made in our understanding with regard to the pathogenesis, diagnosis, staging and the efficacy of management of obstructive jaundice.2 .

Obstructive jaundice of varied etiology is one of the cause s of admission to hospitals across North Karnataka. To diagnose the cause, site of obstruction and management of a case of surgical jaundice is indeed a challenging task for the surgeon. Hence, a comprehensive study of the etiology, clinical presentation a nd management of obstructive jaundice is of paramount importance in the appropriate management of these patients.

OBJECTIVES OF THE STUDY

- To study the clinical history and presentation of obstructive jaundice.
- To study the various causes and sites of obstruction of the biliary tree.
- To study the different modalities of treatment of obstructive jaundice.

REVIEW OF LITERATURE

Jaundice is a generic term, which describe s yellow pigmentation of the skin, mucus membrane or sclera.

Mention of jaundice is made in the works of Hippocrates (400 BC) who pointed out that persistent jaundice may be due to cancer or cirrhosis of liver. 3. Gallstones have been described in Chilean mummies since the second and third centuries AD. Galen in second century AD in his humoral concept of disease attributed abnormalities of yellow bile, black bile, blood and phlegma within the body to cause disease.3,4

- Francis Glisson (1640), Abrahmson vater (1720) and Ruggero Oddi (1887) refined anatomy with description of sphincteric mechanism.3,4

- Charcot (1877), discussed the symptoms associated with the passage of CBD stones which were jaundice, pain abdomen and fever (Charcot triad).

- Telfer Reynold added hypotension and alte red mental status to Charcot's triad
- (Reynold's Pentad) related to sepsis with cholangitis.5

- Langenbunch performed first cholecystectomy in the year 1882.

- Robert Abbe (1889) was the first to perform choledochotomy.

- Lawson Trait performed choledocholithotomy.

- Ludwig Courvoisier (1843-1918) stated Courvoisier's law.3

Courvoisier's law: In obstruction of the common bile duct due to a stone, distension of the gall bladder seldom occurs, the organ usually is already shriveled. In obstruction from other causes, distension is common. If there is no disease of gallbladder and the obstruction is due to a cancer of the ampulla, pancreas or bile duct, then the gallbladder may well be distended.

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- William Stewart Halstead performed choledochoduodenal anastomosis for ampullary carcinoma.

- Emil Theoder Kocher's introduced kocher's incision and kocher's maneuver.5

- Charles McBurney – Transduodenal choledochotomy.

- Hans Kehr – Invented T-tube.3

- John B. Murphy – Cholecystoenterostomy avoiding choledochotomy.

- The first mention of carcinoma of gallbladder was published in 1777 in Ratio
- Mendendi of Maximillian Stall.

- Fredrich discussed carcinoma of gallbladde r and suggested the relationship between gall bladder stone and cancer.3

- Graham Cole (1925) – Oral cholecystography

- Mirrizzi (1931) – Intraoperative cholangiography.

- Okuda (1973) – CHIBA needle for percutaneous transhepatic cholangiography.

- Wildegans of Germany (1953) introduced modern choledochoscope.

- Shore and Shore (1965) – Flexible choledochoscope.

- Yamakawa (1975) – Percutaneous transhepatic cholangioscopy.
- McCune and Oi (1970) – ERCP.
- Kawai et al – Endoscopic papillotomy.
- First laparoscopic CBD exploration by Philips Peterson.

ANATOMY

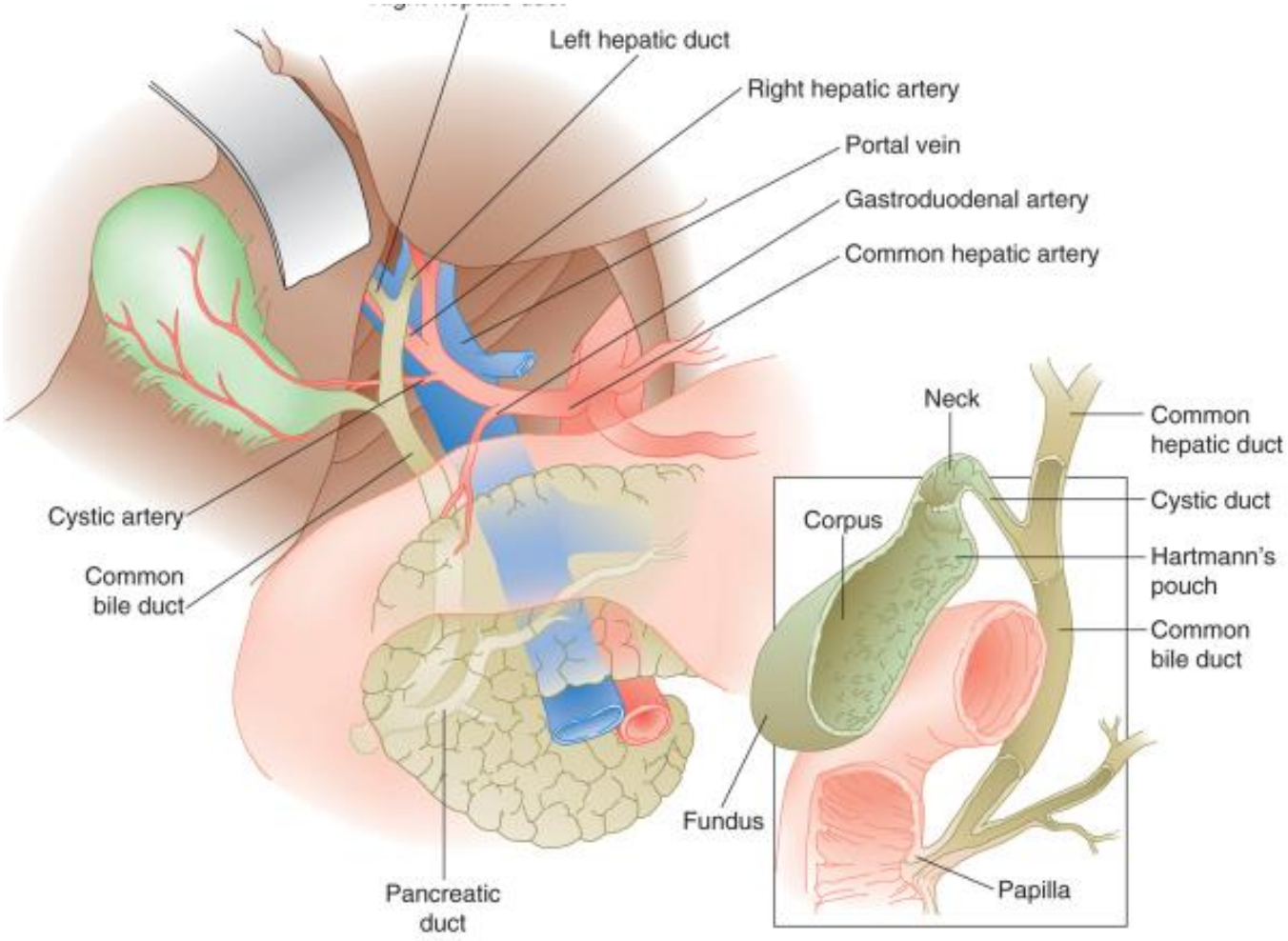
Development

Liver develops from an endodermal bud that arises from the ventral aspect of the gut, at the point of junction between foregut and midgut. The bud enlarges and soon shows a division into:

- Cranial part – Pars hepatica
- Caudal part – Pars cystica

1. Pars hepatica: divides into ri ght and left parts each of which forms one lobe of the liver.

2. Pars cystica: Gives origin to the gall bladder and to the cystic duct. The part of the hepatic bud proximal to the pars cystica forms the bile duct. Bile duct at first opens on the ventral aspect of the developing duodenum. But as a result of differential



growth of the duodenal wall and as a result of the rotation of the duodenal loop, it comes to open on the dorso-medial aspect of the duodenum along with the ventral pancreatic bud.6,7,8

Anomalies of the extrahepatic duct system

A. Abnormal length: Variation in the level at which the various ducts join each other.

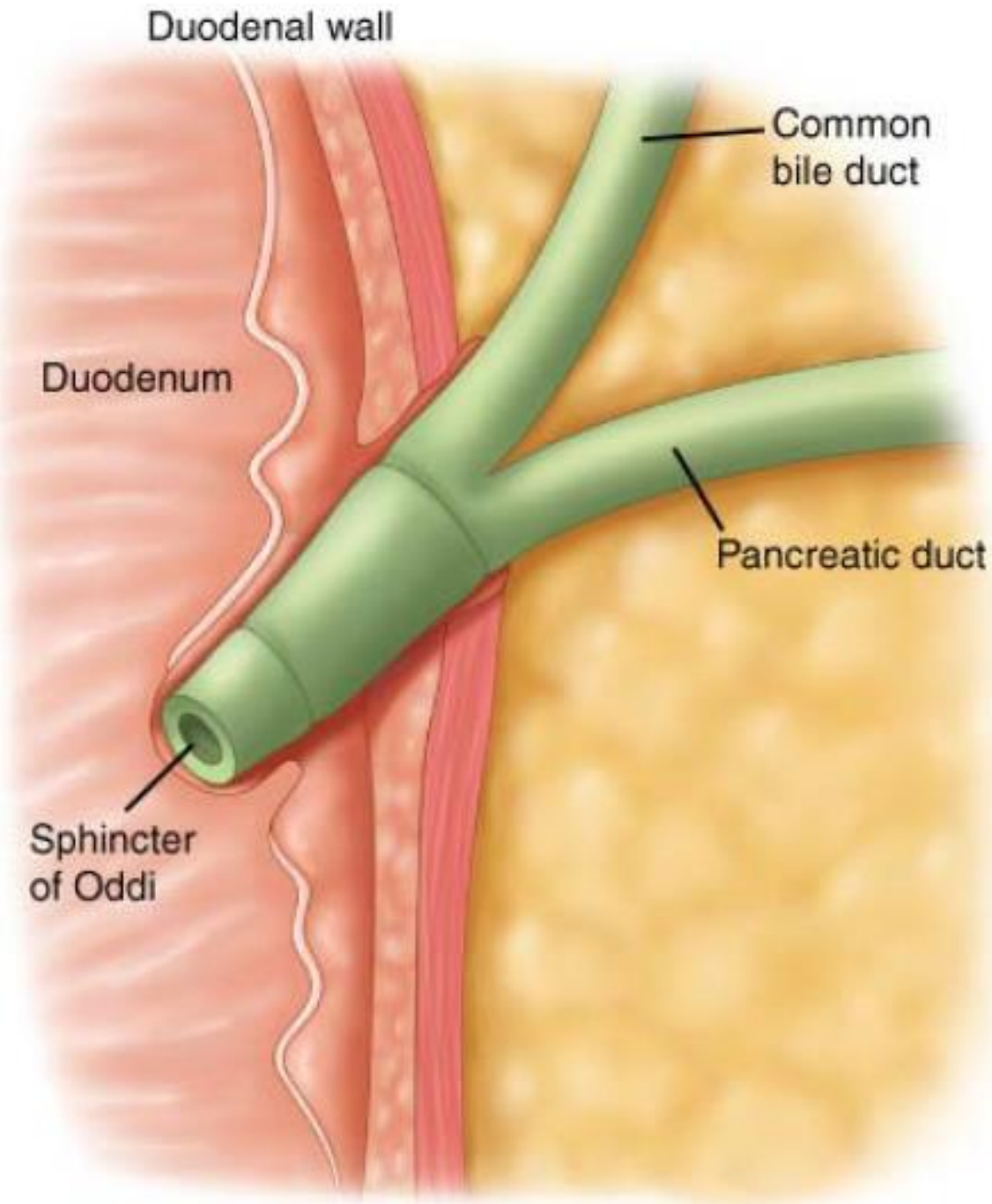
B. Abnormal mode of termination

- Cystic duct may join left side of the common hepatic duct
- Cystic duct may end in the right hepatic duct
- Cystic duct may pass anterior to the duodenum, before joining the common hepatic duct.
- Bile duct may open into the pyloric, or even the cardiac end of the stomach.

C. Atresia: Parts of the duct system and sometimes the whole of it may be absent.

D. Duplication: Parts of duct system may be duplicated.8

Liver is divided into two major portions, the right lobe and left lobe, which are respectively drained by right hepatic and left hepatic ducts. The right and left hepatic ducts converge at the liver hilus to constitute the common hepatic duct. Cystic duct joins common hepatic duct to form common bile duct. The common bile duct courses downwards and backwards anterior to portal vein and lateral to hepatic artery, in the porta hepatis. The common bile duct is divided into four part s: Supraduodenal, Retro duodenal, Para duodenal and Intra duodenal. The retro duodenal portion of the common bile duct approaches the second portion of duodenum obliquely accompanied by the terminal part of duct of Wirsung and opens in the duodenum at papilla of vater.6,9


Duodenal wall
Common bile duct
Duodenum
Pancreatic duct
Sphincter of Oddi

Blood supply

Gall bladder is supplied by cystic artery. The blood suppl y to the biliary ducts is derived from the hepatic, cystic and superi or pancreaticoduodenal arteries. Veins drain directly into liver or form tributaries of the portal vein. Gastroduodenal branches to CBD runs on the side of CBD at 3 ‘0’ clock and 9 ‘0’ clock position. Retro portal artery from celiac plexus runs posterior to portal vein to supply post-aspect of CBD.

Vascular anomalies

(a). Accessory cystic arteries – arising from right hepatic artery.

(b). Accessory cystic artery – arising from left hepatic artery.

(c). Hepatic artery- arising from superior mesenteric artery.

(d). Accessory hepatic arteries arising from the coeliac trunk and Superior mesenteric arteries.

(e). Anterior transposition of right hepatic artery and cystic artery

(f). Recurrent (caterpillar hump) right hepatic artery.

Nerve supply

Consists of sympathetic and parasympathetic fibres passing in the hepatic plexus and being joined at the portahepatis by branches from the anterior vagal trunk.

PHYSIOLOGY

One of the major function liver is to secrete bile normally between 600 to 1200 ml/day.

Bile is secreted in two stages by the liver:

Initially bile is secreted by liver hepatocytes, which is rich in bile acids, cholesterol and other organic constituents, which flow into bile canaliculi.

Next, the bile flows peripherally towards the interlobular septa, where the canaliculi empty into larger ducts, the hepatic duct and common bile duct and then to duodenum or to gall bladder through cystic duct.

Composition of bile

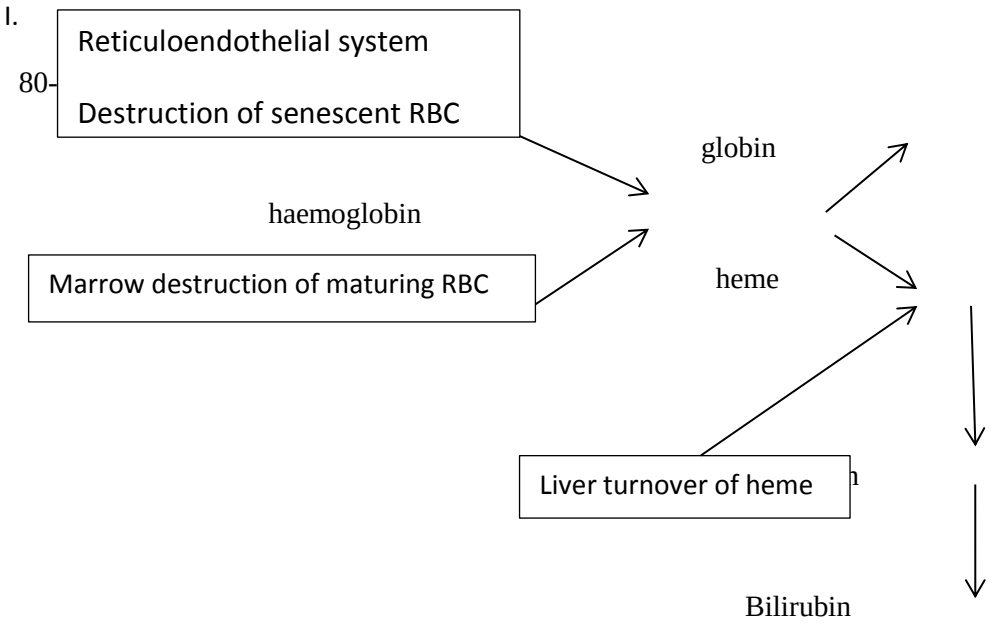
- Water → 97.5 gm/dl
- Bile salts → 1.1 gm/dl
- Bilirubin → 0.04 gm/dl
- Cholesterol → 0.1 gm/dl
- Fatty acids → 0.12 gm/dl
- Lecithin → 0.04 gm/dl
- Na → 145 mEq/L
- K → 5 mEq/L
- Ca → 5 mEq/L
- Cl → 100 mEq/L
- HCO_3 → 28 mEq/L

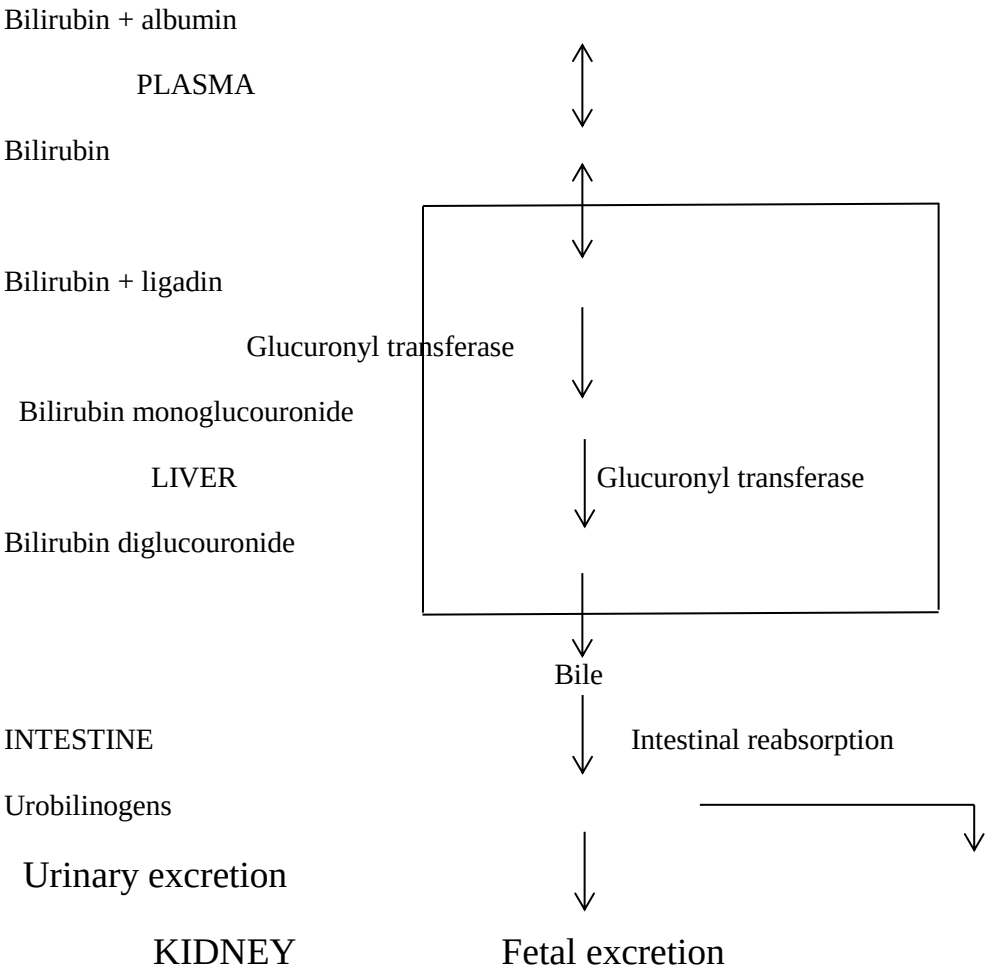
Metabolism

Hepatic metabolism of bilirubin occurs in three phases:

(1) Uptake (2) Conjugation (3) Excretion

Metabolism and Excretion of Bilirubin




Bilirubin + albumin
PLASMA
Bilirubin
Bilirubin + ligadin
Glucuronyl transferase
Bilirubin monoglucouronide
LIVER
Glucuronyl transferase
Bilirubin diglucouronide
Bile
INTESTINE
Intestinal reabsorption
Urobilinogens
Urinary excretion
KIDNEY
Fetal excretion

Surgical Jaundice

A complete or partial obstruction of biliary flow can cause jaundice and this may be intra or extra hepatic.

Causes of surgical jaundice classified into:13

A. In the lumen of duct

- Choledocholithiasis
- Parasitic infestation due to: Hydatid disease, Ascariasis
- Hemobilia

B. In the wall of duct

1. Congenital

- Biliary atresia
- Choledochal cyst

2. Acquired

- Papillary stenosis
- Strictures

a. Post-traumatic

b. Post-surgical

- . Injuries at cholecystectomy

- Exploration of CBD

- Pancreatic operation

- Gastrectomy

- Biliary enteric anastomosis

- Operation on liver and portal vein

c. Post-inflammatory strictures

- Gallstones

- Chronic pancreatitis

- Chronic duodenal ulcer

- Parasitic inflammation

- Recurrent pyogenic cholangitis

d. Primary sclerosing cholangitis

e. Following radiotherapy

f. Mirrizzi’s syndrome

3. Malignant causes

- Ca Gall bladder
- Cholangiocarcinoma
- Ca of ampulla of vater

C. Outside the wall

1. Benign: Pseudocyst of pancreas

2. Malignant:

   - Ca head of pancreas
   - Enlarged lymph nodes at portahepatitis
   - Periampullary Ca
   - Extra biliary malignancy

PATHOPHYSIOLOGY OF BILIARY OBSTRUCTION

Cholestasis is defined as the failure of normal bile to reach the duodenum. It is classified as:

1. Extrahepatic cholestasis

2. Intrahepatic cholestasis

Intrahepatic cholestasis: No demonstrable obstruction to the major bile ducts. They are caused due to drugs, hepatitis, hormones, Primary biliary cirrhosis and septicaemia.

Extrahepatic cholestasis: encompasses conditions where there is physical obstruction to the bile ducts. Cholestasis begins with distension of the bile duct proximal to the obstruction, including intrahepatic duct system. The bile stasis in the duct radicals within the portal triads leads to pro liferation of the epithelial lining cells. Sometimes accompanied by an increase in

surrounding fibrous tissue. Eventually the canaliculi become distended with bile. Distended canaliculi ruptures, which lead to extravasation of bile producing so, called bile lakes surrounded by injured or necrotic liver cells . Because stasis of bile predisposes to ascending bacterial infections, extrahepatic cholestasis may be complicated by cholangitis. Escherichia coli produce beta-glucuronidase, which may lead to deconjugation of bilirubin in bile. This may lead to the formation of primary common bile duct stones with a high bile pigment as contents.

.If obstruction continues, the reticulin laid down in the periportal area matures to hard type, causing fibrosis around the bile duct, which may further aggravate the cholestasis.14,15

Biochemical changes[10 , 16]

Bilirubin

Conjugated hyperbiliurubinaemia is the classic biochemical feature of obstructive jaundice. Conjugated bilirubin is water-soluble and penetration to body fluids is high, thus producing more jaundice than unconjugated pigment.

Alkaline phosphatase

The level rises in cholestasis and to a lesser extent when liver cell are damaged. The mechanisms of the increase are complex. Hepatic synthesis of the alkaline phosphatase by the hepatocytes is increased and this depends on intact protein and RNA synthesis.

Gamma glutamyl transpeptidase (GGT)

Serum values are increased in cholestasis and hepatocellular diseases. Levels parallel serum alkaline phosphatase in cholestasis and ma y be used to confirm that a raised serum phosphatase is of hepatobiliary origin. It is also elevated in alcohol intake, pancreatitis, chronic lung disease, renal failure and congestive heart failure.

Protein synthesis

The liver occupies a central role in protein synthesis and albumin is quantitatively the most important of plasma protein formed in th e liver. The relatively long half-life of serum albumin (20 days) makes the serum albumin

level a better index of severity and prognosis in patients with chronic liver disease. Alteration in serum albumin levels may reflect not only disturbances in synthesis but also changes in the rate of catabolism, dilution by expanded plasma volume or enhanced loss from the gastrointestinal tract and kidneys. The most important aspect of protein synthesis relates to the liver in the maintenance of the normal blood coagulation process.

Clotting factors

Liver disease is a common cause of impaired coagulation. Normal serum activities of the vitamin-K dependent coagulation factor proenzymes (factors II, VII, IX and X), as assessed by the one stage prothrombin time, depend on both intact hepatic synthesis and adequate intestinal absorption of vitamin K. In cholestatic disease prolonged prothrombin time can be improved by parenteral administration of vitamin K. A characteristic pattern of abnormalities occurs in patients with severe liver dysfunction; it consists of low plasma fibrinogen level, a prolonged prothrombin time, and a normal or prolonged partial thromboplastin time.

ETIOPATHOGENESIS

In the lumen of duct

A. Choledocholithiasis

Nearly all calculi found in the common b ile duct were originally formed in the gallbladder and migrate down the cystic duct in to the common bile duct. Calculi in common bile duct can be divided into: [15, 17]

1. Primary common bile duct stones
2. Secondary common bile duct stones

1. Primary common bile duct stones[18, 19]

These stones are formed primarily in the CBD and do not originate from gallbladder. These are caused due to bile duct stasis or du e to infection. Almost all calculi are pigment stones. They are solitary, ovoid, light brown in colour, soft and easily crushable.

Disturbance in the flow of bile as well as introduction of infected material into the biliary tract, may be associated with previous operation that disturb the usual motility dynamics of the sphincter, such as sphincterotomy, sphincteroplasty and biliary enteric anastomosis and also condition that obstruct the flow of bile into duodenum such as sphincter fibrosis, chronic pancreatitis and periampullary duodenal diverticula can lead to stasis of bile resulting in bile duct calculi and infection of biliary system.

2. **Secondary common bile duct stones**

Are those that have migrated into the biliary system from the gall bladder.

3. Retained stones in common bile duct are those that present at some point in time following cholecystectomy with or without concomitant bile duct stone.

B. **Hemobilia**

Hemobilia is a rare cause of upper GI bleeding that most often results from blunt or penetrating hepatic injury, with fistula formation within the liver between a vascular structure and the biliary duct system. Jaundice occurs due to

acute extrahepatic biliary obstruction owing to the blood clot formation in the common bile duct. Non-traumatic cause of hemobilia includes hepatic abscesses, choledocholithiasis or oriental cholangiohepatitis.

C. Parasitic infestation of bile duct

1. Ascariasis: It is the most common helminthic infestation of bile ducts, caused by Ascariasis lumbricoides. In the presence of massive duodenal infestation the worms can enter the biliary system.[20]

2. **Clonorchiasis**: It is endemic to Asia and is ca used by the liver fluke, clonorchis sinensis, which is transmitted by ingestion of infected raw fish. The intrahepatic ducts are the natural habitat, where they cause obstruction, periductal inflammation and fibrosis.[21]

2. Echinococcus granulosus (Hydatid cyst) : It is the usual organism responsible for hydatid disease. Large cysts in the liver may compress the intrahepatic biliary radicals and rupture into the bile duct and may re lease daughter cysts causing obstructive jaundice and fibrotic change in the biliary tree.[22]

In the wall of duct

1**. CONGENITAL**

a. Biliary atresia

The occurrence of biliary atresia is embryological. Failure of vacuolization of the solid embryonic bile ducts filled by proliferating epithelial cells was supposed to produce this malformation. Probably only a small percentage of cases are congenital malformations or intrauterine catastrophies.23

Types

Type I: Atresia of CBD, with a common hepatic duct remnant.

Type II: Atresia of common hepatic duct and bile duct with right and left duct remnants.

Type III: Atresia of the extrahepatic ductal system.

b. Choledochal cyst[24, 25]

It is defined as an isolated or combined congenital dilatation of the extrahepatic and intrahepatic biliary tree. Mostly confined to extrahepatic biliary tree starting from bifurcation of right and left hepatic duct to the opening of pancreatic duct. Three theories have been stated for the formation of the cyst.

- Anomalous pancreatic duct and biliary duct junction – causes pancreatic reflux into common bile duct, leading to high-pressure in common bile duct.
- Abnormal canalization of the bile duct during embryogenesis, with distal obstruction causing weakening of bile duct wall.
- Abnormality of autonomic innervation of the extrahepatic tree.

Caroli's Disease

It is a rare, congenital, non-familial condition, characterized by multiple sacular dilatation of intrahepatic ducts separated by segments of normal or

stenotic ducts. Usually associated with congenital hepatic fibrosis, medullary sponge kidney, cholangiocarcinoma and stone formation.[24, 25]

Todani's Classification of Choledochal Cysts

Type I – Dilatation of the extra-hepatic biliary tree, a-cystic, b-focal, c-fusiform

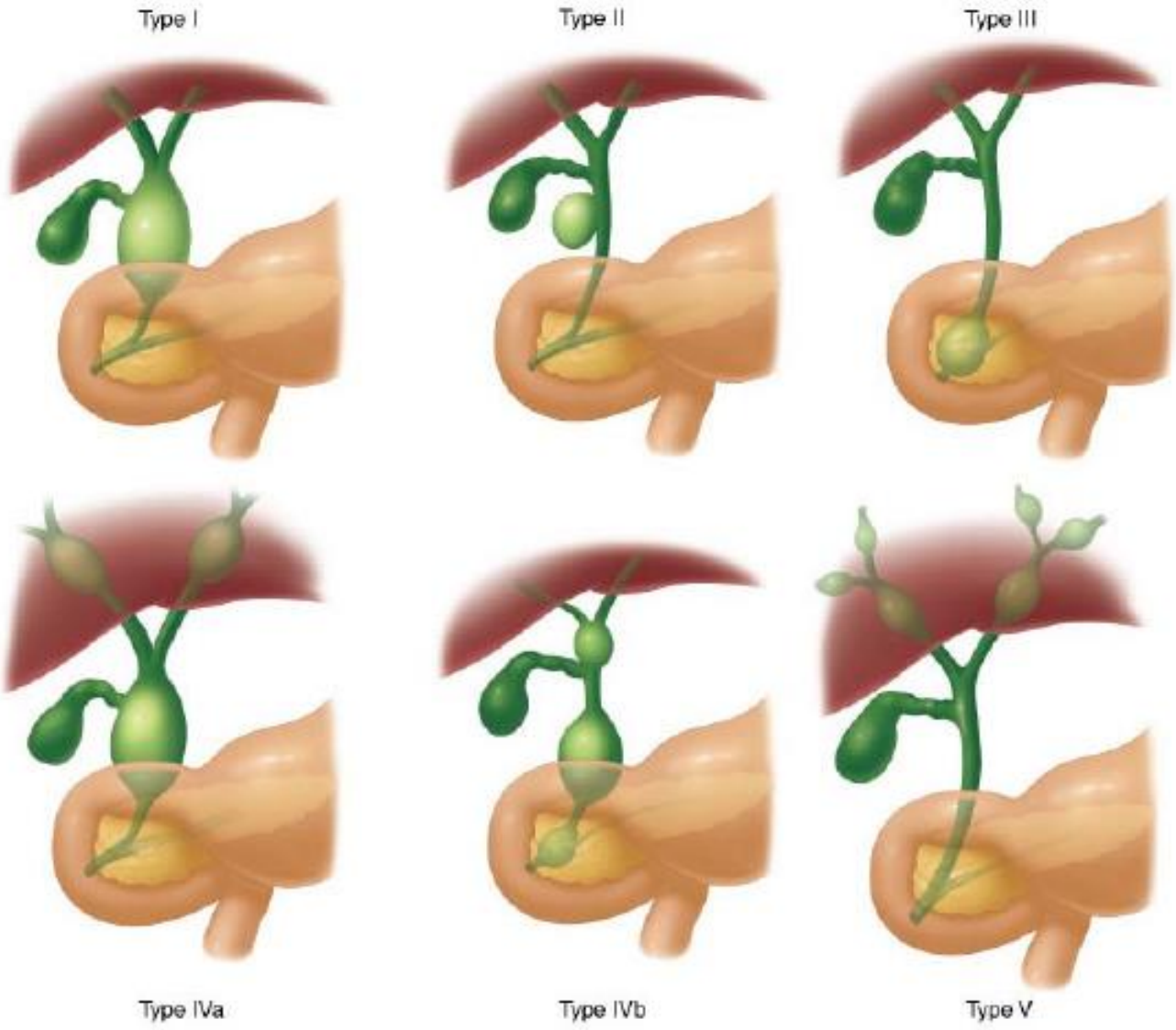
Type II- Saccular diverticulum of extrahepatic bile duct

Type III- Biliary tree dilatation within the duodenum: choledochocele

Type IVa- Dilatation of intra and extra-hepatic biliary tree

Type IVb- Multiple extrahepatic cysts

Type V-Dilatation of intrahepatic ducts


Type I
Type II
Type III
Type IVa
Type IVb
Type V

2. ACQUIRED

Papillary stenosis: It has been defined as an obstructive disease of the papilla, which is organic and benign. It is usually primary, of unknown pathogenesis or secondary to inflammation such as duodenal ulcer or pancreatitis or passed out common bile duct calculi.[26]

Benign biliary strictures

Benign stenosis and strictures of the bile ducts occurs in number of conditions and may affect intrahepatic or extrahepatic biliary tree.

Causes

A. BILE DUCT INJURIES[27-30]

I. Postoperative bile duct strictures

1. Cholecystectomy and exploration of common bile duct
2. Other operative procedures
    - Biliary enteric anastomosis
    - Operation of liver or portal vein
    - Pancreatic operation
    - Gastrectomy

II. Stricture after blunt or penetrating injury

B. POST-INFLAMMATORY STRICTURE WITH [27,30]

1. Cholelithiasis/Choledocholithiasis

2. Chronic pancreatitis

3. Chronic duodenal ulcer

4. Abscess or inflammation in sub hepatic region

5. Parasitic infection

6. Recurrent pyogenic cholangitis

C. **Primary Sclerosing Cholangitis**

D. Radiation-induced Cholangitis

E. Papillary Stenosis

Postoperative bile duct strictures

The great majority of injuries to the bile duct occur during cholecystectomy with or without exploration of the CBD. They also occur in other operation on either the stomach, pancreas or liver or during surgeries for portal hypertension.[32]

Number of factors relate to bile duct injury with cholecystectomy[29-32]

A. Anatomical Variation

There are wide anatomical variations in extrahepatic biliary tree and adjacent hepatic artery and portal venous structure. Anomalies of the vessels are in the tune of 20% and hence very common. The most common for right hepatic artery to arise in whole or in part from the superior mesenteric trunk. The important ductal anomalies are related to the manner of confluence of right and left hepatic ducts and of the cystic duct with the common hepatic duct and bile duct anomalies mentioned earlier.

B**. Use of diathermy near Calot's triangle**.

C. **Technical factors**: Bile duct injuries occur following cholecystectomy performed by surgeons who are inadequately trained or inexperienced. Traction on the gallbladder while applying clips on the cystic duct ma y include a part of the CBD wall which leads to stricture formation. The hepatic duct or common bile duct is often assumed as the cystic duct and has been excised.

D. **Attempts to control haemorrhage during cholecystectomy**: There may be damage to the bile duct if clamps are applied blindly.

E**. Bile duct ischaemia**: Bile duct blood supply runs in three columns, one posterior and two lateral. It is suggested that damage to these vessels may result in ischaemia to the bile duct, with consequent necrosis and stricture.

F. **Pathological factor**: Acute cholecystitis may be accompanied by extensive edema in the region of porta hepatitis and Calot's triangle and there may be considerable friability during dissection.[28]

Primary sclerosing cholangitis

It has an unknown etiology. It is a progressive cholestatic disorder characterized by a fibrosing inflammatory process, which affects the intrahepatic and or extrahepatic ducts. It may occur alone or in association with inflammatory bowel disease. The strictures are typically short and annular

alternating with normal or minimally dilated segments leading to characteristic beaded appearance.

Classification of bile duct stricture [32](Bismuth Classification)

Type I: Low common hepatic duct stricture; hepatic duct stump > 2 cm.

Type II: Mid common hepatic duct stump < 2 cm

Type III: High stricture (Hilar)

Type IV: Destruction of hilar confluence

Type V: Involvement of sectoral right branch alone or with common duct

III. **Malignant causes**

a. **Ca Gallbladder**[35,36]

This tumor represents only 2% of all cancers , but it is commonest site of cancer in biliary tract.

Etiology

75-98% of patients with gallbladder Ca has gallstones and also common in the presence of chronic cholecystitis. Others include Porcelain gallbladder (10-25% risk) Choledochal cyst, inflammatory bowel disease, polyposis coli and Primary sclerosing Cholangitis.

Pathology

85% of gallbladder carcinoma is adenocarcinoma, other variants common are mesenchymal tumors, carcinoid, lymphoma, squamous and adenosquamous carcinoma.

TNM staging for gallbladder Ca

	Tumor	**Nodes**	**Metastasis**
Stage 0	Tis	N_0	M_0
Stage 1	T_1	N_0	M_0
Stage 2	T_2	N_0	M_0
Stage 3	T_3	N_0	M_0
Stage 4	T_{1-3}	N_1	M_0
Stage 5	T_4	Any N	M_0
Stage 6	Any T	Any N	M_1

Tis: Ca in situ

T: Tumor limited to mucosa or muscularis

T1: Tumor invades perimuscular connective tissue or serosa

T2: Tumor invades liver (< 2 cm) or one adjacent organ.

T3: Tumor extends > 2 cm into liver or two or more adjacent organ.

Nevin classification for staging for gallbladder carcinoma

Depth of tumor

Stage I: Mucosa

Stage II: Muscularis

Stage III: Serosa

Stage IV: Liver invasion

Stage V: Adjacent organ or distant metastasis

b. **Cholangiocarcinoma**[38,39]

The incidence of bile duct tumors increases with age. Has even distribution between men and women. The most common is adenoc arcinoma (95%) and remaining are squamous, leiomyosarcoma, mucoepidermosis, carcinoid and cystadenocarcinoma.

Causes of cholangiocarcinoma are Primary sclerosing cholangitis, choledochal cyst, liver fluke infestation, CBD stone, thorotrast and asbestos exposure.

TNM staging for Extrahepatic Cholangiocarcinoma[40]:

T1: Tumor limited to mucosa/muscle

T2: Tumor invades periductal tissue

T3: Tumor invades adjacent structure

	Tumor	**Nodes**	**Metastasis**
Stage IA	T_1	N_0	M_0
Stage IB	T_2	N_0	M_0
Stage IIA	T_3	N_0	M_0
Stage IIB	T_{1-3}	M_0	M_0
Stage III	T_4	Any N	M_0
Stage IV	Any T	Any N	M_1

c. **Periampullary carcinoma**[41]

Periampullary cancers include a group of malignant neoplasms arising at or near the ampulla of vater, within 2 cms of radius fr om ampulla. Most of them are adenocarcinoma arising from the head of pancreas (60%), ampulla of vater (20%), distal common bile duct (10%) or second part of duodenum (10%).

Risk factors [41]

- Cigarette smoking
- Diet – rich in animal fat
- Chronic pancreatitis
- Post Gastrectomy, cholecystectomy
- Chemical exposure: naphthylamine, benzidine
- Hereditary: Familial polyposis, Gardener's syndrome
- Diabetes mellitus

d. **Ca head of pancreas**

Accounts for 60% of periampullary carcinomas. Atleast 2/3 rd of cases of pancreatic cancer arises in the head of the gland. Ductal carcinoma of the pancreas accounts for more than 90% of all malignant pancreatic exocrine

tumours. Other variants are giant cell carcinoma, adenosquamous carcinoma, mucinous carcinoma and acinar cell carcinoma.

Pancreatic cancer has a propensity for perineural invasion within and beyond the gland and for rapid lymphatic spread. The commonest sites for extralymphatic involvement are the liver, peritoneum and lungs.

e. **Carcinoma of Ampulla of Vater**

Accounts for 20% of periampullary carcinomas. Most are adenocarcinomas. Though a number of other tumor histologies such as carcinoids, other neuroendocrine tumors and sarcomas may arise. Spread of the tumors is by local extension to involve the pancreas and duodenum and metastasis to regional lymph node.

CLINICAL FEATURES[44]

Abdominal pain

Typically, the pain is felt in the right uppe r quadrant or epigastrium, with frequent radiation to the intrascapular area and typically lasts for 30 minutes to several hours.

The pain due to bile duct obstruction is due to distension and increased pressure within the bile duct. With cholangitis both the pain and the initial phase of fullness and discomfort are produced at lower intraluminal pressure.

It is inflammatory or malignant lesions spreads to the surface of the liver or gall bladder "somatic" pain results.

Jaundice

Jaundice is the abnormal accumulation of bilirubin in body tissue, which occurs when the serum bilirubin level exceeds 50 mol/L. Excess bilirubin, causes a yellow tinting to the skin, sclera and mucous membranes. Jaundice is important feature of disease in the blood, liver or biliary system.

Pruritus

Pruritus is an important symptom of liver disease. It lasts longer than 3 to 4 weeks regardless of the cause. It tends to be most marked on the

extremities, is present less often on the trunk, rarely on the neck and face. It is often more troublesome after a hot bath and at night, when the skin is warm. The pruritus of liver disease has been attributed to the high plasma concentration of bile salts.

Nausea, vomiting

Nausea and vomiting is often a striking feature in patients with acute biliary obstruction but may not be present.

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Anorexia

Anorexia is a common symptom of liver disease, particularly in jaundiced patients with either hepatocellular failure or biliary obstruction. Anorexia may be profound. Weight loss is probably due to anorexia and reduced food intake.

Bowel functions

A moderately increase in stool frequency w ith passage of soft or loose stools due to increased fecal fat resulting from a lowered intraluminal concentration of bile salts. It is an uncommon presenting symptom except in malignant biliary obstruction – steatorrhoea occurs with biliary obstruction but much higher level of fecal fat results when pancreatic duct is blocked.

Stools and urine

Fecal colour gives a good indication whether cholestasis is total, intermittent or decreasing. Occult blood in stools – sign of malignancy.

Bile salts, deficient in the intestine in cholestasis are essential for the normal colour of the stools. It is usually pale in colour in cholestasis.

Urine is dark in colour in cholestatic jaundice because of increase in circulating conjugated bilirubin.

Mass per abdomen – Gall bladder palpable or Courvoisier's gall bladder.

Bleeding

Patients may complain of spontaneous bleeding from the nose and gums or of easy bruising, because the prothrombin time is prolonged due to decrease in vitamin K absorption.

Fever

This is secondary to acute cholangitis, which results from two factors, obstruction of the biliary tree and bacteria in bile.

General Physical Examination

- A parous middle-aged obese female is a candidate for gallstones while chances for cancerous biliary obstruction increases with age

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- Widely set eyes, a prominent forehead, flat nose and small chin are features of any form of persistent intrahepatic cholestasis in childhood

  .

- Xanthoma and Xanthelesma around the eyes suggests chronic cholestasis. Elevated plasma cholesterol levels are seen as de posits in palmar creases, below the breast, chest or back. The tuberous lesions appear later and are found on extensor surfaces, especially the wrists, elbows, knees, ankles and buttocks.
- Fever may be present in metastatic liver disease and with primary carcinoma of pancreas and stomach.
- Dyspnoea and tachypnoea is commonly seen due to ascitis, abdominal organomegaly due to elevated and restricted movement of the diaphragm.
- Jaundice: Yellowish discolouration of sclera, skin and mucous membrane due to increase in serum bilirubin above 2 mg/dl. Patients with prolonged biliary obstruction have a deep greenish hue compared

with hemolytic jaundice where it is mild yellow and in hepatocellular jaundice where it is orange.

- An increase in melanin pigmentation is seen with many chronic liver disease and chronic cholestatic disorders.
- Scratch marks result from severe pruritus.
- Spider naevi, excessive bruising, tiny petechial haemorrhages indicates the presence of chronic liver diseases.

INVESTIGATIONS

Laboratory Examinations [44-46]

1. **Urine bilirubin**

When bilirubinuria is present the urine is unusually dark brown in colour. Test strips can detect as little as 1 to 2 μmol of bilirubin per litre. Bilirubinuria occurs even with small increase in plasma-conjugated bilirubin and usually precedes jaundice.

Absence of bilirubinuria is important in a jaundiced patient, as it suggests an unconjugated hyperbilirubinemia or hemolysis.

2. Urinary Urobilinogen

Bilirubin esters entering the intestine undergo bacterial hydrolysis and degradation in the ileum and colon with the production of urobilinogen. Normal levels in the stools – 40 to 200 mg/24 hrs.

Urinary urobilinogen give a reaction with Ehlirich's aldehyde reagent. A normal value is 0 to 4 mg/24 hrs.

In the presence of liver damage more urobilinogen escapes hepatic uptake and biliary excretion and it is excreted in urine.

3. Liver Function Tests

a. Serum bilirubin

Normal values: Total - upto 1.2 mg/100 ml

Direct - upto 0.4 mg/100 ml

Indirect - 0.4 to 0.8 mg/100 ml

The standard test for bilirubin is the Vandenberg reaction. The basis for this colorimetric test is the differing solubility of conjugated and unconjugated bilirubin.

Hepatocellular disease impairs excretory function of bile to a greater degree than the ability to conjugate bilirubin and therefore the hyperbilirubinemia in this setting is predominantly of the conjugated type. It is very difficult to distinguish between hepatocellular disease and extrahepatic biliary obstruction solely on the basis of conjugated hyperbilirubinemia.

In patients with jaundice secondary to extr ahepatic obstruction, the determination of the direct fraction of bilirubin is more sensitive index than total bilirubin.

b. **Alkaline Phosphatase**

Normal – 3 to 13 King Armstrong (K-A) units (Or)

1.5 to 4 Bobansky units (or)

35 – 130 IU/L

The level of alkaline phosphate rises in cholestasis and to a lesser extent when liver cells are damaged. The rise in alkaline phosphatase along with increase in Gamma glutamyl transpeptidase is specific for hepatobiliary origin

c. Gamma Glutamyl Transpeptidase

Serum values are increased in cholestasis and hepatocellular diseases. Levels parallel to serum alkaline phosphatase in cholestasis and may be used to confirm that a raised serum phosphatase is of hepatobiliary origin.

d**. Aminotransferases**

1**. Serum Glutamic Oxaloacetic Transaminase (SGOT) or Aspartate Transaminase (AST)** [46]

This is a mitochondrial enzyme present in large quantities in heart, liver, skeletal muscles and kidney and the serum level increases whenever these tissues are acutely destroyed, presumably due to release from damaged cells.

Normal: SGOT: 6-40 units (Karmen)

5-40 IU/ml

0-15 IM/ml

2. **Serum Glutamic Pyruvic Transaminase (SGPT) or Alanine Transaminase (ALT)**

This is a cytosolic enzyme also present in liver. Compar ed to SGOT major amount is present in liver. Therefore, this is more specific for liver damage than SGOT. Normal: SGPT: 6-36 units (Karmen)

0-15 IM/ml

5-35 IU/L

3. **Albumin**: Normal – 35 to 50 g/L

Synthesized in the hepatocytes. Its half-life is 15 to 20 days. Hypoalbuminaemia reflects severe liver damage and decrease albumin synthesis. Other causes for hypoalbuminaemia are:

- Protein losing enteropathies
- Nephrotic syndrome
- Chronic infection

4. **Globulin**: Normal – 5 to 15 g/L

They are a group of protein mostly ma de up of gamma globulin, produced by B- lymphocytes. Alpha and beta globulin are produced in hepatocytes. Globulin increases in chronic liver disease.

5. **A/G ratio**: 1.5-3/1. Any change or reversal in the ratio indicates liver damage.

6. **Prothrombin time**: Normal – 12 to 16 seconds which collectively measures factors II, V, VII, and X. Biosynthesis of these depends on Vitamin K, Prothrombin time may be elevated in hepatitis, cirrhosis and obstructive jaundice. Marked prolongation of prothrombin time > 5 sec above control is a poor prognostic factor.

Radiological studies[47-51]

1. Ultrasound

Ultrasound examination of the hepatobiliary system is an important first line, non- invasive investigation. Patient preparation for ultrasound should include fasting for 12 hours. Dilated bile ducts, gall bladder disease, hepatic tumours and some diffuse hepatic abnormality are identified.[48,49]

Normal ultrasound shows the liver to have mixed echogenicity. Portal and hepatic veins, inferior venacava and aorta are shown. Normal intrahepatic ducts measuring 1 to 3 mm in diameter and common bile duct 2 to 7 mm in diameter are seen on ultrasound.

Ultrasound plays a vital role in the evaluation of focal liver disease, screening for liver metastasis, hepatocellular carcinoma, portal hypertension, surgical obstructive jaundice and hepatic veno-occlusive disease.

Sonography is ideally suited to study the internal architecture of a focal mass and distinguish a solid from a cystic lesion. The addition of colour Doppler flow imaging further helps in characterizing mass lesions and assessing patency of vessels.

The major drawback of this technique is that it is highly operator dependent.

2. Computed Tomography (CT)

CT also shows dilated bile ducts distinguishing obstructive from non-obstructive jaundice in 90% of cases. But as a screening procedure it has no advantage over ultrasound. It is however, more likely than ultrasound to show the level and cause of obstruction.

Advancement of CT technology including the development spiral scanners and more recently, multi-detector row CT scanners and the development of three dimensional (3D) imaging software have significantly improved the ability of CT to image patients with obstructive biliopathy.[52]

3. Endoscopic retrograde cholangiopancreatography (ERCP)

With the help of ERCP, diseases of the oesophagus, stomach, duodenum, pancreas and the biliary tract including duodenal diverticula and fistulae may be diagnosed.[53]

ERCP is performed with a side viewing endoscope, either video or fibre-optic. The stomach and duodenum are inspected and biopsy and cytology

specimen taken if indicated. The papilla is identified. The cannula is then introduced under direct vision into the papilla and contrast injected under fluoroscopic control. X-ray films are taken. The success rate of ERCP is 80 to 90%, but depends on experience.[54]

Indication

- Used to show duct strictures
- Gall bladder and common bile duct stones
- Pancreatic and bile juice may be obtained for culture, aspiration cytology
- After biliary surgery
- Pancreatic disease
- Cytology or biopsy from malignant growth or strictures

Complication

- Acute pancreatitis
- Cholangitis

4. **Percutaneous transhepatic cholangiography**

Contrast is injected percutaneously into a bile duct within the liver. The procedure is done in the radiology department. The "skinny" chiba needle is 22 G, is introduced in the 7th, 8th, or 9th right intercostal space at mid axillary line, with the help of USG & the contrast is injected. Biliary tree is identified and if any dilated ducts are encountered, they should be catheterized and external or internal biliary drainage established.[55]

The technique is easy and the success rate is 100% if intrahepatic bile ducts are dilated.

Indications

- When ERCP has failed.
- Endoscopic access is difficult (Hepaticoenterostomy, Billroth II).
- Brush cytology and biliary biopsy may be performed.

Complication

- Bleeding
- Biliary peritonitis
- Septicaemia

5. **Magnetic resonance cholangiopancreatography (MRCP)**

MRCP shows water containing bile and pancreatic juice within bile duct and pancreatic duct without the need for injection of contrast. MRCP is more expensive than USG and CT, and is not available in all hospital.

It has an overall accuracy of greater th an 90% in showing common bile duct stones. Highly accurate in showing bile duct stricture and pancreatic carcinoma.

6. **Endoscopic ultrasound (EU)**

This is done using an endoscope which has a miniature ultrasound transducer mounted at its tip. Most endoscopes used for ultrasonography have a mechanical rotating scanner at the tip and are side or oblique viewing. Recognizing the structures seen at endoscopic ultrasonography requires a sufficient period at training and this has limited its general availability to specialist centres.

In the hepatobiliary system its prominent role is in the detection and evaluation of pancreatic tumours. It also detects common bile duct stones and can be used for image- directed biopsy. Accuracy of endoscopic ultrasound for choledocholithiasis is greater than 90%

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7. Biliary scintigraphy[59]

The technetium-labelled iminodiacetic acid derivative (IDA) is cleared from the plasma by hepatocellular organic anion transport and excreted in the bile. The method may be used to determine patency of the cystic duct in suspected acute cholecystitis. The gall bladder ejection fraction can be calculated. Cholescintigraphy can show whether the bile duct is obstructed. Scintigraphy is also useful in assessing the patency of biliary- enteric anastomosis and may show biliary leaks after cholecystectomy or liver transplantation.

8. **Operative and postoperative cholangiography**

They are indicated when there is stones present in common bile duct. After exploration of the common bile duct, cholangiography should always be performed, using high kilovolt peak technique and full strength contrast.

Any debris may cause filling defects less sharply defined than those caused by gallstones. Any obstruction of the common bile duct, there will be no flow contrast into the duodenum.

Postoperative cholangiography using contrast injected gently should be undertaken routinely before final removal of a T-tube draining the biliary tree.

It is essential to obtain filling of all right and left intrahepatic radicals, the common hepatic duct and common bile duct and flow to duodenum before removal of T-tube.

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9. **Abdominal Radiograph**

The abdominal radiograph may be performed as part of the initial surgical evaluation of the patient presenting with an abdominal condition.

- 10 to 15% of gallstones will be visualized by this examination.
- Gas in biliary tree (Aerobilia) may be seen after endoscopic sphincterotomy or surgical bile duct or bowel anastomosis.

10. **Barium contrast upper gastrointestinal X-ray**

X-rays can distinguish between a neoplastic and calculus obstruction. Early changes in malignancy include short thick mucosal folds in duodenum with relative stasis. The circumscribed filling defect in the gastric silhouette described as PAD SIGN, the post-bulbar impression of duodenum due to a dilated common duct in Ca pancreas. The reversed "3 sign" of Frostberg in periampullary carcinoma are all important radiological signs of malignancy.

11. **Oral Cholecystography**

Agent used usually is Iopanoic acid. The material is transported in the blood bound to albumin and selectively taken up by the liver, where it is conjugated and excreted as the glucuronide of Iopanoic acid. The contrast material then enters the gall bladder and is concentrated there. Gallstones detected in most of the patients and anomalies of gallbladder visualized.

Non-visualization of gall bladder has numerous causes including failure to ingest the agent, intestinal obstruction preventing passage through the small bowel, malabsorption, liver dysfunction and biliary tract abnormalities preventing flow of contrast into the gall bladder.

12. **Intravenous cholangiography**

From the time of its development in 1953 until recently, intravenous cholangiography using sodium or meglumine iodipamide has been regarded as an important technology for examination of the biliary tree. It has been reported that the common duct will be visualized in 90% of patients with normal levels of bilirubin. Problems with this procedure include faint visualization, morbidity and mortality associated with the contrast agent and non- visualization in the jaundiced patient.

Despite these problems, it has been a widely used procedure especially for evaluating the patient who has undergone cholecystectomy for biliary leak.

As the error rate is rather high and there are other imaging technique that offer better visualization of biliary tree the value of intravenous cholangiography is doubtful.

Preoperative preparation

- Obesity increases the technical difficulties for the surgeon and makes post-operative complications more likely. Weight reduction under the control of dietician if possible.
- Use of contraceptive pills adds the risk of venous thrombosis and it is advisable that OCP be stopped prior to and after 6 weeks of surgery.
- An abnormal prothrombin activity increases the risk of haemorrhage. Administration of Vitamin K dose will reduce this risk.
- Pre-operative antibiotic prophylactic is given.
- Insertion of Nasogastric tube, intravenous fluid infusion and urinary catheter to monitor output is a must.
- The patient's blood grouping in case of transfusion is necessary.
- Nutritional status should be assessed and improved if necessary.
- The renal, cardiovascular system and CNS should be evaluated before surgery.

- Jaundice patient have a high-risk of postoperative renal failure which can be reduced by operating during diuresis produced by Mannitol with fluid supplementation.

Anaesthesia

General anaesthesia for biliary tract surgery is essentially no different from that of any intraabdominal operation. However, there are a few points of particular interest to the anaesthetist.

- The presence of abnormal liver function test s requires caution to be taken according to the degree of abnormality with the dosage of all drugs used, as almost all depend on the liver for their detoxication. Halothane is contraindicated in the presence of abnormal liver function.
- General anaesthesia must produce sleep, analgesic, good muscle relaxation and stable blood pressure.
- Care must also be taken during the operation to avoid kinking of inferior vena cava during deep retraction which results in drop in cardiac output.
- Intravenous atropine 0.6 mg given first before the operative cholangiogram helps to diminish any spasm of the sphincter of oddi.

Position of patient

Patient is placed on the cassette table top. Patient is placed supine with a foam pillow is inserted under the ankles to raise the calves off the table. Another foam pillow is inserted at back of the patient to elevate the gall bladder bed.

Incision

- Kocher's right subcostal
- Midline incision
- Right upper paramedian

Surgical procedures

In current surgical practice, various operative procedures have been performed for obstructive jaundice, depending on the cause. The choice of procedure also depends on the experience and preference of the surgeon.

- Cholecystectomy with common bile duct exploration with stone
- Removal/dilatation/ sphincteroplasty and T-tube drainage.
- Cholecystectomy with Choledochoduodenectomy with T-tube drainage.
- Cholecystojejunostomy
- Pancreaticoduodenectomy (Whipple's procedure)
- Palliative operation for relieving obstructive jaundice due to malignant disease.
- Pancreaticojejunostomy
- Non-surgical biliary drainage

Different operations for biliary stricture

A. Exploration of the common bile duct [61 ,62]

The operation of choledochotomy carries a mortality of atleast four times greater than cholecystectomy alone.

Indications

- History of jaundice
- Multiple small stones or Single faceted stone in the gall bladder
- A dilated cystic duct
- Induration of head of the pancreas
- Dilated common duct – more than 8 mm
- A palpable stone in the common bile duct during surgery.

Operation

Opening of the duct

Stay sutures of catgut are inserted into the common duct near either side of the anterior aspect about 2 cm above the first part of the duodenum and a longitudinal incision is made with a fine knife.

Exploration (Distal)

Fogarty catheter is inserted down the common bile duct into the duodenum and the balloon inflated. The catheter is gently withdrawn until it is halted by the sphincter of Oddi. The lower end of the duct is palpated and stone felt against the catheter, above the balloon. A bulldog clamp is placed across the common duct above the opening to prevent any stones escaping into the proximal ducts. The balloon is deflated and the catheter gently brought through the sphincter this can be traced by the palpating fingers. The balloon is

reflated and steadily withdrawn bringing the stone with it. This procedure is repeated until no more stones are withdrawn.

Exploration (Proximal)

The catheter is reinserted into the proximal segment and the procedure is repeated in the left and right hepatic ducts. The bulldog clip is placed on the common duct below the opening to prevent stones falling into the distal duct. The balloon is inflated until some resistance is felt and withdrawn with the pressure maintained on the syringe. This is necessary because the lumen of the duct increases in diameter and unless the balloon continues to fill the lumen, a stone may slip past.

Assessment by X-ray

The catheter is inserted into the common duct so that the balloon lies just distal to the opening where it is initiated to occlude the duct. After checking th e position of the x-ray machine, about 10 ml of hypaque is injected an d films are taken. The balloon is deflated and the procedure repeated with the catheter in the common hepatic duct. For fixed stone bougie's or Desjardin's forceps are used to dislodge the stone and withdrawn.

Closure of the duct can be done with or without a T-tube. Catgut is used and usually it is continuous.

Gall bladder is removed, cystic duct, cystic artery ligatures are checked. Any bile leak from the common bile duct is inspected.

Removal of T-tube postoperatively

The t-tube is allowed to drain freely into a bile bag for 5 days when it is clipped for 1 hour after meals. This is increased by 1 hour each day so that by the tenth day the tube is clipped all day. A T-tube cholangiogram is obtained a final check that the duct system is normal and if so the skin suture is withdrawn and the tube removed.

D. **Sphincterotomy**[63]

Sphincterotomy requires duodenotomy placed at the level of the sphincter of Oddi, division of the sphincter and suture of the wall of common

bile duct to the duodenum. It has advantages of facilitating inspection of the pa pilla, biopsy and pancreatic radiography, if required.

Indication

- Common bile duct stone
- Stricture at the lower end of the common bile duct

E. Exploratory choledochoscopy[64]

The choledoscope permits visual inspection within the bile ducts during surgical exploration for gallstones and may greatly facilitate the exploration of the common and hepatic bile ducts and the localisation and retrieval of stones.

Position of patient

The operating table should have facilities for x-ray to enable the operative cholangiography to be done. The patient should be positioned with a few degrees of feet down and lateral tilt to the right.

Incision

A transverse right upper abdominal incision.

Operation

Once the abdomen is opened, the duodenum and the hepatic flexure of the colon are retracted downwards. A clear exposure of anterior aspect of the supraduodenal common bile duct is done. The choledochotomy should be placed as low as possible, above the superior border of the duodenum.

The choledochoscope is introduced into the common bile duct in a distal direction. The interior of the common bile duct is inspected as the instrument is advanced distally. A gallstone may be retrieved from the common bile duct under direct vision. A fine balloon catheter (Fogarty) is passed down the channel of the choledochoscope and passed beyond the stone and inflated. The balloon, stone and instrument all withdrawn together.

For multiple mobile stones in the duct are tractable wire basket may be passed down the choledochoscope channel. The basket should be used to open as a dragnet to avoid crushing of the stones.

Proximal choledochoscopy is done to view hepatic ducts. Choledochotomy is closed with 3/0 catgut, either interrupted or continuous.

F. **Transduodenal exploration of the bile duct (Biliary Sphincterotomy)**[63]

Indication

- Impacted stone to the lower end of the common bile duct.
- Stenosis of the papilla and sphincter
- Re-exploration

Contraindication

- A single large stone in the supraduodenal portion of the common bile duct that does not descend to the sphincteric region.
- Multiple facetted stones locked in the bile duct.
  - Long stricture of terminal common bile duct
  - Presence of acute pancreatitis.

Operation

Either a right paramedian or Kocher's subcostal incision provides good access to the biliary tree once the abdomen is opened, the hepatic flexure of the colon and proximal transverse colon are mobilized and reflected caudally exposing the head of the pancreas and the duodenal.

Head of the pancreas and duodenum are mobilized forward and medially. Dissection continues until the aorta is visualized and the third part of the duodenum is free. Papilla is located on the medial wall of the duodenum. A

small bulldog clip is then placed across the supraduodenal part of the common bile duct in order to prevent calculi slipping back into the common hepatic duct and its tributaries.

The duodenal walls are incised longitudinally or transversely. Babcock tissue forceps are applied to the longitudinal fold, distal to the papilla and the latter is drawn into duodenotomy incision. A grooved director or lacrimal probe is passed into the papillary orifice and hence into the common bile duct.

Sphincterotomy is done at 10’0 clock position. Stones are extracted with Desjardins forceps and Fogarty balloon catheters. Clearance of the duct is confirmed by intraoperative post-exploratory cholangiography.

Alternatively, choledochoscopy may be used. The duodenotomy is closed in its original axis using a continuous suture (catgut) and fine non-absorbable Lambert suture.

Complication

Bleeding: Persistent bleeding of the sphincterotomy incision usually occurs from a divided circumferential duodenal artery which is best secured by suture.

Cholangitis: It is important to remove all calculi and provide an adequate sphincterotomy with free drainage.

Acute pancreatitis: Probing into pancreatic duct should be avoided and to ensure that no suture encircle the pancreatic duct.

G. **Duodenoscopic sphincterotomy for removal of duct stones**[65, 66]

Fibreoptic oesophagogastroduodenoscopy is now a routine procedure. ERCP is performed by passing a Teflon catheter through the biopsy channel of the duodenoscope and placing it directly in the orifice of the papilla of vater under direct vision. Contrast material is then injected during fluoroscopy and appropriate radiographs are ta ken. The technique is performed under sedation.

Sphincterotomy

After endoscopic cholangiogram has demonstrated stones, the Teflon catheter is replaced in the distal bile duct by a diathermy wire.

After confirming its position radiographically the wire is withdrawn slightly and made taut to produce a bow, pressing on the root of the papilla.

Diathermy current is applied to produce a cut 15-20 mm long. The aim is to ablate the biliary sphincters and a view directly up the bile duct is obtained.

Stone extraction

After sphincterotomy, some allow stones to pass spontaneously (< 1 cm in diameter). Few prefer to remove all stones using balloon catheters or wire basket.

Complication

- Bleeding (mainly from the sphincterotomy site)
- Cholangitis
- Pancreatitis
- Retroperitoneal perforation

Endoscopic treatment for patients without stones

Patient with convincing biliary symptoms following biliary surgery are suspected to have sphincter of Oddi dysfunction or stenosis; this may result from the passage of stones or instrumentation.

Endoscopical sphincterotomy is a logical and effective form of treatment when functional obstruction is present.

For malignant biliary obstruction, when the patient unfit or unsuitable for surgery, jaundice can be relieved by performing sphincterotomy through the tumour or by placing a prosthetic stent.

Palliative operations for jaundice due to malignant disease[67, 68]

Indications

Non-resectable carcinoma of the pancreas and peri-ampullary carcinoma

Two symptoms are readily palliated by operation: duodenal obstruction and jaundice.

For duodenal obstruction gastroenterostomy with entero-enterostomy is done.

For jaundice

a. **Cholecysto-enterostomy**

Indication: Tumour must atleast be 5 cms below the junction between the cystic duct and the common bile duct.

Procedure

- Gall bladder emptied using a trocar suction apparatus.
- Jejunal loop is laid alongside the gall bladder of cholecyst oenterostomy done using 2/0 catgut.

b. **Choledochojejunostomy**

Indication

- Distal common bile duct malignant stricture
- Malignant growth distal common bile duct/ Peri-ampullary carcinoma

End-to-side anastomosis

The common duct is dissected free and transected, and the distal end is oversewn. The proximal end is then sutured to the loop of jejunum. The opening in the jejunal loop, made at the apex should be a little smaller than the diameter of the common duct.

Anastomosis can be completed with or without T-tube.

Side-to-side anastomosis

If the bulk of the jejunum a llows, then a series of accu rate interrupted all coats sutures is placed between the opening of the common duct and the opening in the jejunum. Anastomosis completed with or without T-tube.

C. **Choledochoduodenostomy** [69]

Indication

- Dilated common bile duct containing infected bile and biliary sludge.
- Multiple intrahepatic stones
- Suppurative cholangiohepatitis

Contraindication

- In patient with a bile duct of normal dimension.

Operation

Incision: Kocher or Paramedian

Exposure: First and second parts of duodenum with addition of the lesser sac and the free edge of the lesser omentum and the liver.

Dissection

The common bile duct is then opened and a specimen of bile sent for culture. Stones are removed and the duct is cleared by copious lavage with saline. The duodenum is freely mobilized by Kocher's maneuver in order to allow it to roll upwards over the anterior surface of the bile duct.

After the duct has been dissected a vertical incision 2-5 cm long is made on its supraduodenal portion and the duodenum is opened in such a way to allow the stoma to be made without tension. The anastomosis is done with interrupted 3/0 chromic catgut.

I. Benign biliary stricture[70]

Diagnosis and Principles of treatment

Damage to the bile duct may be recognized immediately (at operation), early postoperative period or late after discharge.

Immediate repair of bile duct injury

The form of repair will depend upon the se verity and site of the damage, but results are excellent.

a. **Common duct laceration without tissue loss**

Insertion of T-tube through the laceration and closure of the wound with interrupted absorbable sutures. T-tube is removed after 10 days, after cholangiogram.

b. **Common duct laceration with minimum tissue loss**

Loss of part of the wall of common duct occurs when the cystic duct is torn at its origin. Its tissue loss is minimal; the edges are trimmed and closed transversely. T-tube is positioned in common bile duct through a separate incision and removed after four weeks.

End-to-end anastomosis

A clear division of common duct without tissue loss may be repaired by direct anastomosis at the two ends with interrupted sutures over a T-tube, which should be left in situ for three months.

Biliary intestinal anastomosis

Accidental removal of a whole segment of common bile duct will necessitate some form of biliary-intestinal anastomosis using a Roux loop at jejunum.

Late repair of bile duct stricture

1. **Biliary intestinal anastomosis for low bile duct strictures**

Treatment of a stricture at the lower end of the common bile duct will depend upon its site and relationship to the ampulla.

a. Choledochoduodenostomy

To avoid leaving an undrained sump the common duct above the stricture is anastomosed side-to-side to the second part of the duodenum establishing a wide stoma.

b. **Choledochojejunostomy**

Anastomosis of the common duct to a jejunal Roux-en-Y is theoretically more desirable because food particles are less likely to enter biliary tree and obstruct its lumen.

Biliary intestinal anastomosis for mid duct strictures

Anastomosis of dilated common hepatic duct above the stricture to a jejunal Roux-en-Y loop using interrupted catgut. A T-tube inserted into the common hepatic duct with one arm through the anastomosis should remain in situ for three months.

Biliary-intestinal anastomosis for high duct stricture

- The track, which will lead to the mucosa-lined ducts, is first explored with a fine probe , it is then enlarged with Baker's dilators.

- Once a mucosa-lined duct has been identified a rubber catheter is positioned and cholangiogram performed. This will demonstrate anatomy, degree of dilatation and any stones present.

Positioning of a transhepatic tube: Fully curved Randall's forceps are now inserted into the common hepatic ducts and passed along the dilated intrahepatic duct system, usually of the left lobe and then to the periphery. Then tip of forceps grasp the end of the latex rubber tube and is drawn along the ducts to appear at the porta hepatitis.

Construction of Roux loop and exposure of the jejunal mucosa:

- A standard ante- colic jejunal Roux-en-Y is fashioned and the end of the free limb closed in two layers. Just proximal to this closed end a 2 cm diameter disc of seromuscular wall is removed to expose the intact jejunal mucosa.
- The end of the transhepatic tube passed to the jejunal lumen and anchored.
- Traction on the transhepatic tube from above will draw the mucosa, into the lumen of the common duct to create sutureless anastomosis with mucosa-to-mucosa opposition.
- Few interrupted catgut sutures are inserted between the jejunal serosa and liver capsule to maintain the graft in position.

- The upper end of the transhepatic tube is brought out through a separate stab incision in the abdominal wall and fixed securely to the skin with non-absorbable suture.

Bilateral mucosal grafts

For very high stricture involve the carina or the right and left ducts individually. In this event both ducts intubated and the transhepatic tubes inserted separately through the exposed jejunal mucosa to create an epithelial bridge between the two ducts.

Management of the transhepatic tube and vacuum drain

- First 3 postoperative days – low-pressure suction (Robert's pump at 5 cm Hg).
- Later replaced by plastic bag into which bile drains freely.
- On the tenth day cholangiogram to confirm patency of the anastomosis.
- End of the transhepatic duct closed with a spigot.
- Patients are taught to irrigate the tube daily with 20 ml sterile water to maintain patency.
- Three months later the transhepatic tube is removed.

J. Alternative methods of biliary decompression

1. **Intrahepatic cholangiojejunostomy (long wire operation)**: Involves the anastomosis of an exposed peripheral intrahepatic duct to a defunctional limb of jejunum.

2. Percutaneous transhepatic drainage.

Malignant biliary stricture[70]

Proximal tumours

- Hepatic bifurcation tumours
- Hepatic duct tumours

a. Parts to be excised are (radical excision)

- Gall bladder
- Entire supraduodenal common hepatic duct system as far as the right and left hepatic ducts.

b. Join the several hepatic duct openings for two biliary enteric anastomosis

c. Silastic drains through the substance of liver is brought to the inferior surface of the liver.

d. Roux-en-Y loop is constructed and sutured to the jejunum with silastic tube inside (internal) and the other end brought outside the abdominal wall. The tubes are removed after 2-3 months after cholangiogram.

Distal tumours

Tumours of the bile duct below the cystic duct are treated with pancreaticoduodenectomy.

- Whipple's surgery
- Pylorus preserving pancreaticoduodenectomy
- Total pancreatectomy

Non-surgical biliary drainage

- External drainage
- Internal drainage

External drainage

Indications

- Drainage done preoperatively to reduce ope rative mortality and morbidity, especially acute renal failure.
- Incase of acute cholangitis because later surgery is expected to be safer.
- For patients who are inoperable, long term drainage being performed to relieve symptoms of cholestasis.

Prerequisite

Intrahepatic ducts have to be dilated.

Coagulation studies should be normal.

Lateral X-ray screening facilities available.

Procedure

As per percutaneous transhepatic cholangiogram and then a stent is left (j=shaped/pig-tailed catheter placed) for decompression.

Internal drainage

- ERCP and stenting – explained earlier.
- PTC where the guide wire is passed beyond the stricture and then the stent passed. After the insertion of the stent through the stricture, guide wire removed allowing internal decompression of the cholestasis.
- Palliative operation for jaundice due to malignant disease

Pancreatoduodenectomy (Whipple's Operation)

The purpose of the operation of pancreatoduodenectomy is to remove the tumour en bloc with adjacent lymph nodes. The head of the pancreas, the duodenum, the pylorus and distal half of stomach, the gallbladder and th e lower end of the common duct and to restore biliary-pancreatic and gastrointestinal continuity.

Pancreatoduodenectomy is the treatment of choice for operable malignant tumours of the ampulla of vater, the lower end of the common bile duct, the duodenum and the periampullary region of the head of the pancreas.

Incision

- Right Mayo-Robson
- Bilateral subcostal incision

Procedure

I. Retraction of common bile duct: Exposure and ligation of right gastric and gastroduodenal arteries.

II. Finger dissection between pancreas and portal vein from above and finger dissection between pancreas and superior mesenteric vein from below. If both index finger meet, lifting the pancreas forward and then resection can be carried out.

III. Division of stomach (Partial gastrectomy).

IV. Division of common bile duct with the gallbladder.

V. Division of the pancreas in front of the portal vein.

VI. Division and mobilization of the jejunum.

VII. Detachment of duodenum and uncinated process of pancreas from superior mesentric vessels.

VIII. Anastomosis of bile duct to jejunum. Anastomosis of pancreas to jejunum gastrojejunostomy.

Complication

Disruption of pancreatojejunal anastomosis.

Postoperative hemorrhage from gastrointestinal tract.

Pylorus – Preserving Pancreatoduodenectomy

A modification to Whipple's surgery is by preserving stomach and pylorus. The duodenum is transected, usually 3 x 4 cm distal to the pylorus and re-anastomosed to the jejunum at the point where a gastrojejunostomy would have been do ne in the standard operation. The remaining aspects of the procedure are no different.

METHODOLOGY

Source of Data

Patients admitted to the institute of General Surgery at the Rajiv Gandhi government general Hospital, Chennai were taken up for the study. Numbers of cases studied were 50 from

Method of Collection of Data

After admission to RGGGH, a detail ed clinical history and examination of the patient was done. Relevant investigations were undertaken to make a diagnosis. Patients were assessed preoperatively for the fitness for surgery and later subjected to curative or palliative surgery depending on the stage of the disease and general condition of the patient. The resected tissue was subjected to histopathological examination. Postoperatively, patients' condition was assessed and complications were documented. Photographic documentation has been done wherever possible.

Inclusion criteria

Age – More than 12 years.

Patients proved to have obstructive jaundice by any investigative modality during the study period from April 2004 to March 2005.

Exclusion criteria

- Age less than 12 years
- Medical jaundice
- Cases of obstructive jaundice who are unfit for interventional treatment
-

Statistical methods

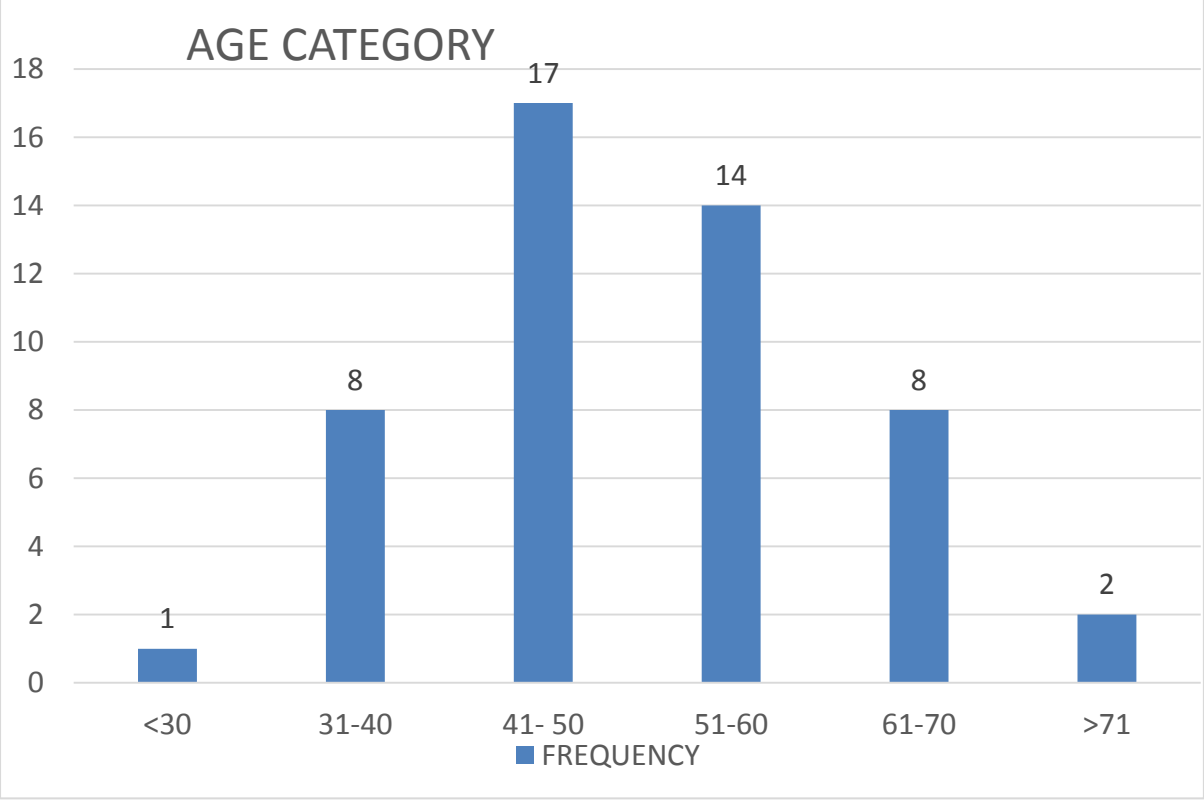
Chi-square and fisher exact test have been used to find the significance of proportion of symptoms &signs between benign and malignant cases. Student t test has been used to find the significance of mean difference of lab parameters between benign and malignant cases. The odds ratio has been used to find the strength of relationship between symptoms & signs of benign and malignant cases. If p value was <0.05 the probability was considered to be statiscally significant.

RESULTS AND OBSERVATIONS

A Prospective clinical study consisting of 50 cases of Obstructive jaundice was undertaken to investigate the pattern of clinical presentation & lab parameters to study the cause of obstructive jaundice and the different modes treatment adopted.

AGE AND SEX DISTRIBUTION

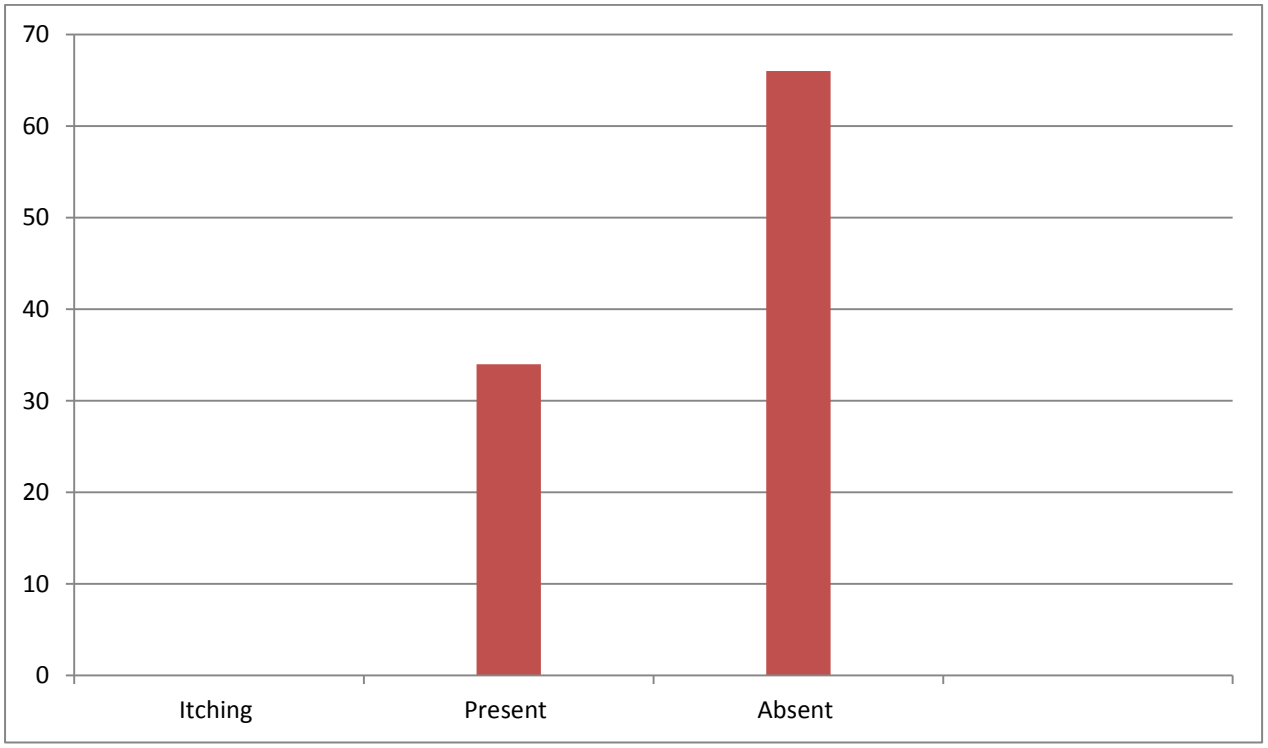
SEX	FREQUENCY	PERCENTAGE (%)
Male	30	60
Female	20	40



Mean age with SD :	51.72 (10.5)	
Mean bilirubin with SD	12.3 (8.6)	

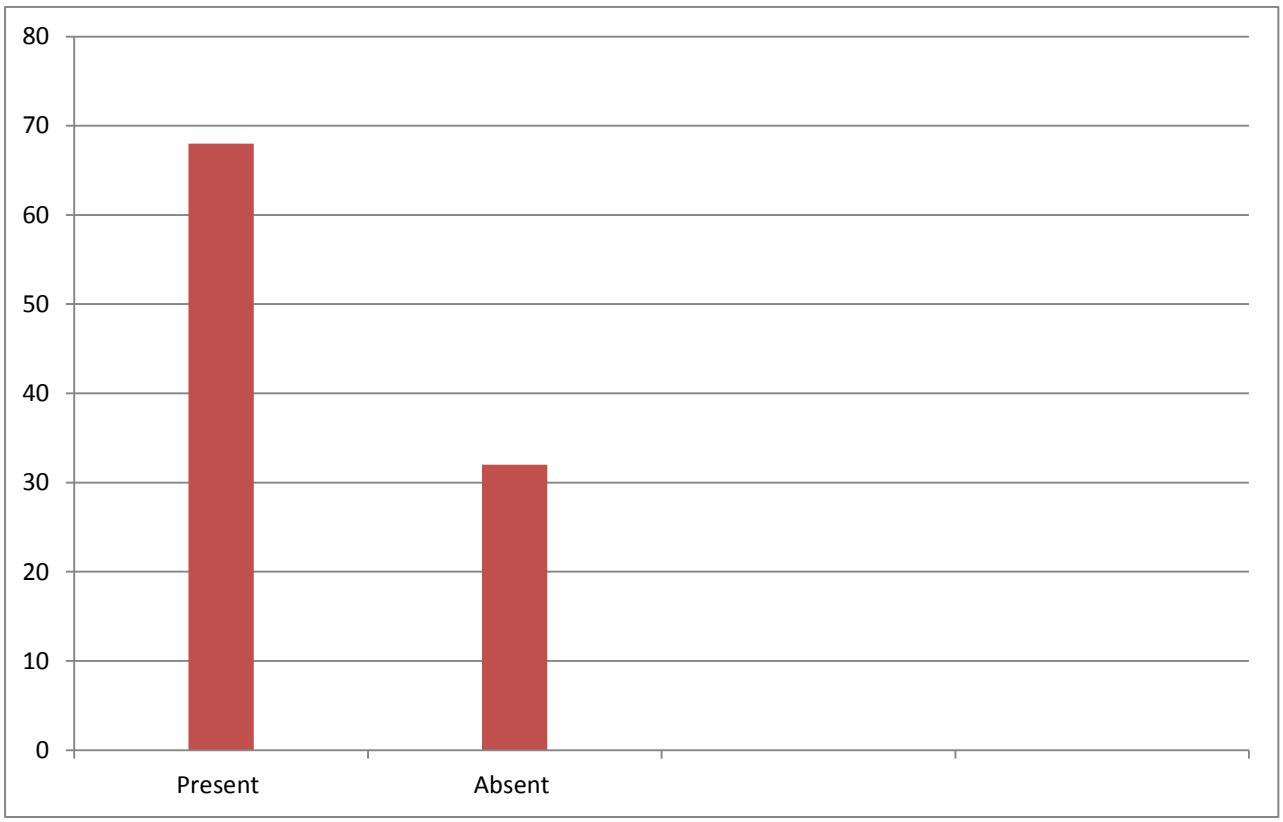
PATIENTS PRESENTING WITH ITCHING AS PRIMARY COMPLAINT

Itching	FREQUENCY	PERCENTAGE (%)
Present	17	34
Absent	33	66



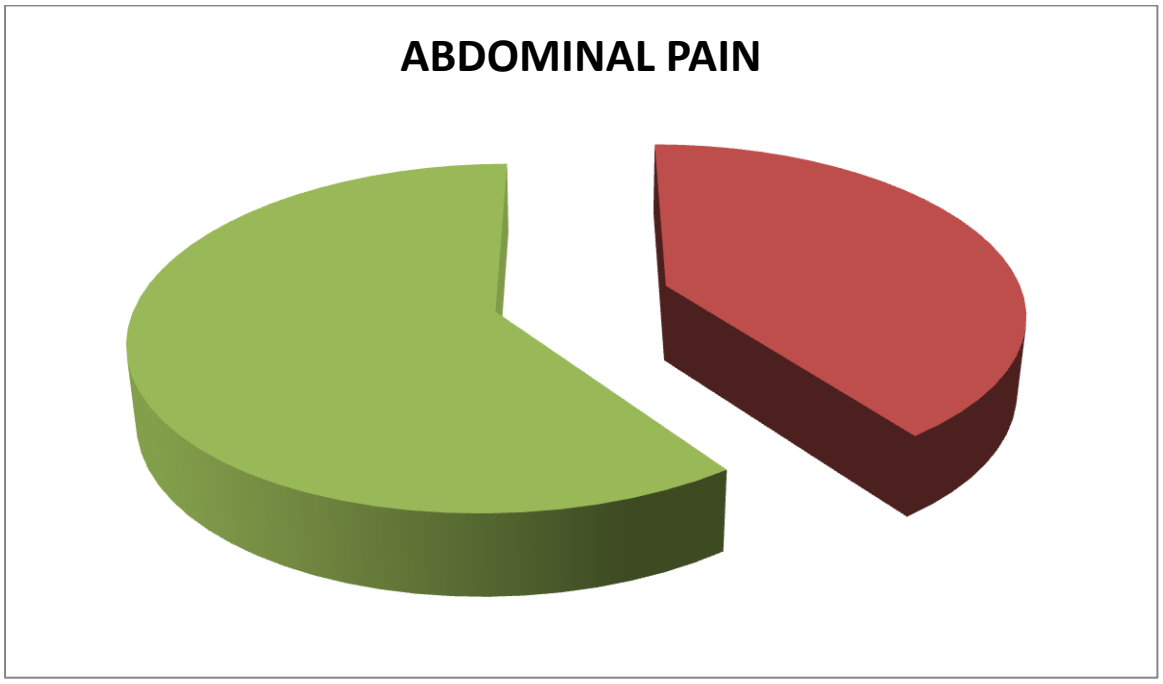
PATIENTS PRESENTING WITH JAUNDICE AS PRIMARY COMPLAINT

Jaundice	FREQUENCY	PERCENTAGE (%)
Present	34	68
Absent	16	32



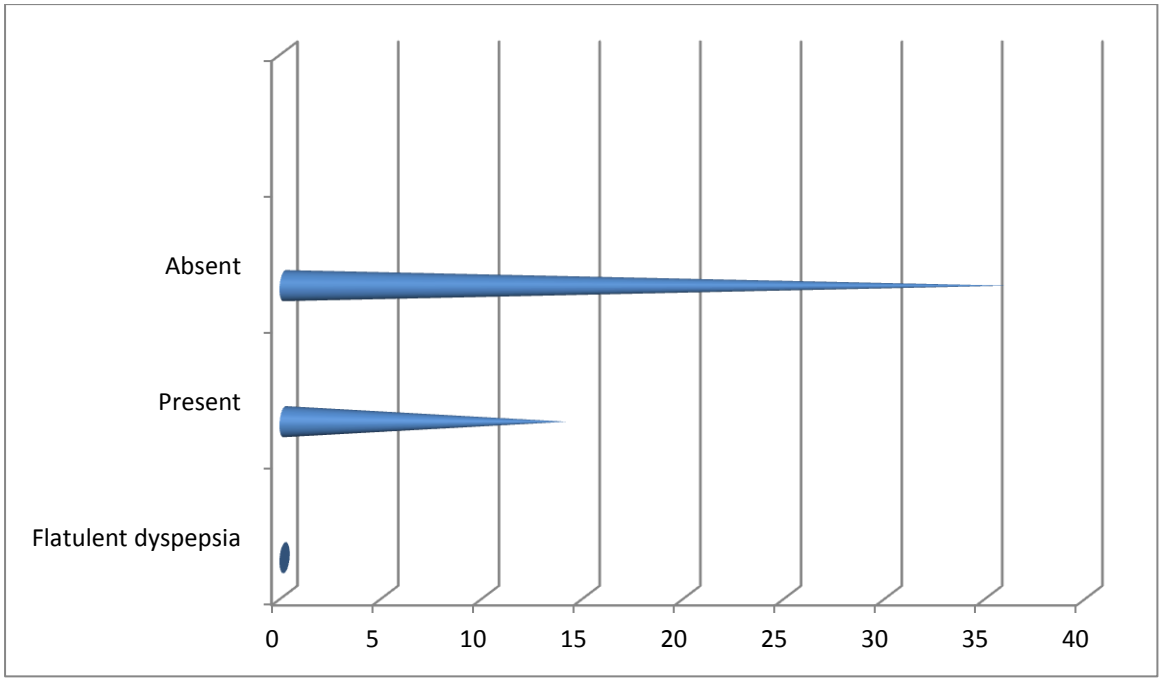
PATIENTS PRESENTING WITH ABDOMINAL PAIN AS PRIMARY COMPLAINT

Abdominal pain	FREQUENCY	PERCENTAGE (%)
Present	20	40
Absent	30	60



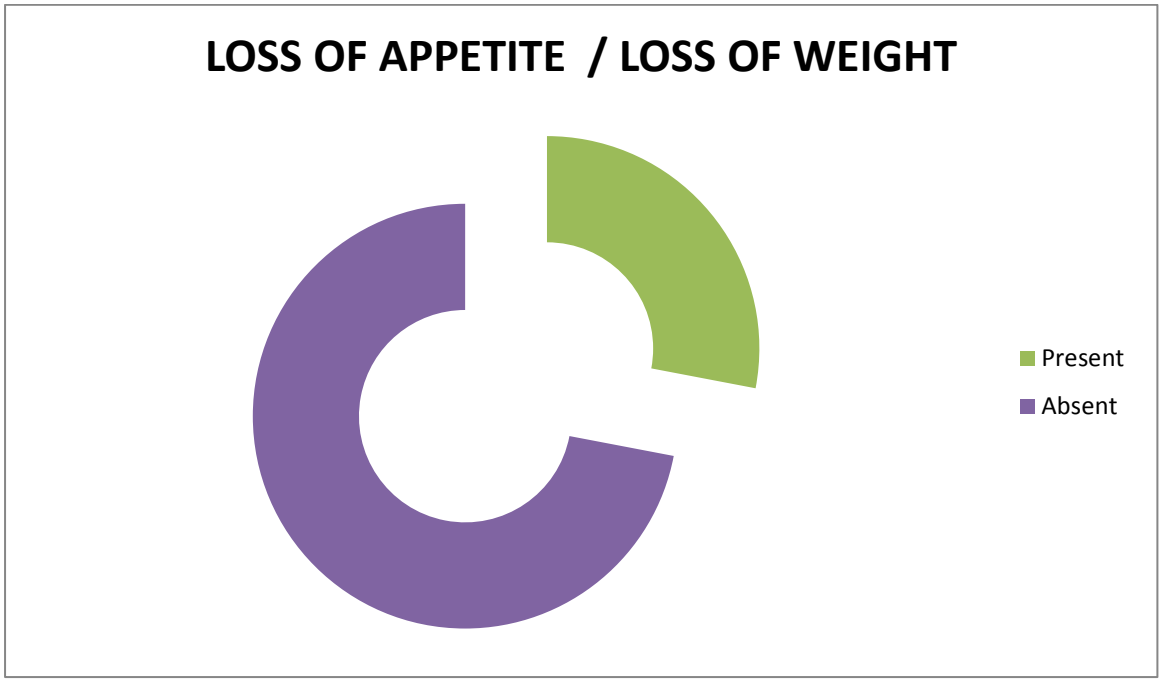
PATIENTS PRESENTING WITH FLATULENT DYSPEPSIA AS PRIMARY COMPLAINT

Flatulent dyspepsia	FREQUENCY	PERCENTAGE (%)
Present	14	28
Absent	36	72



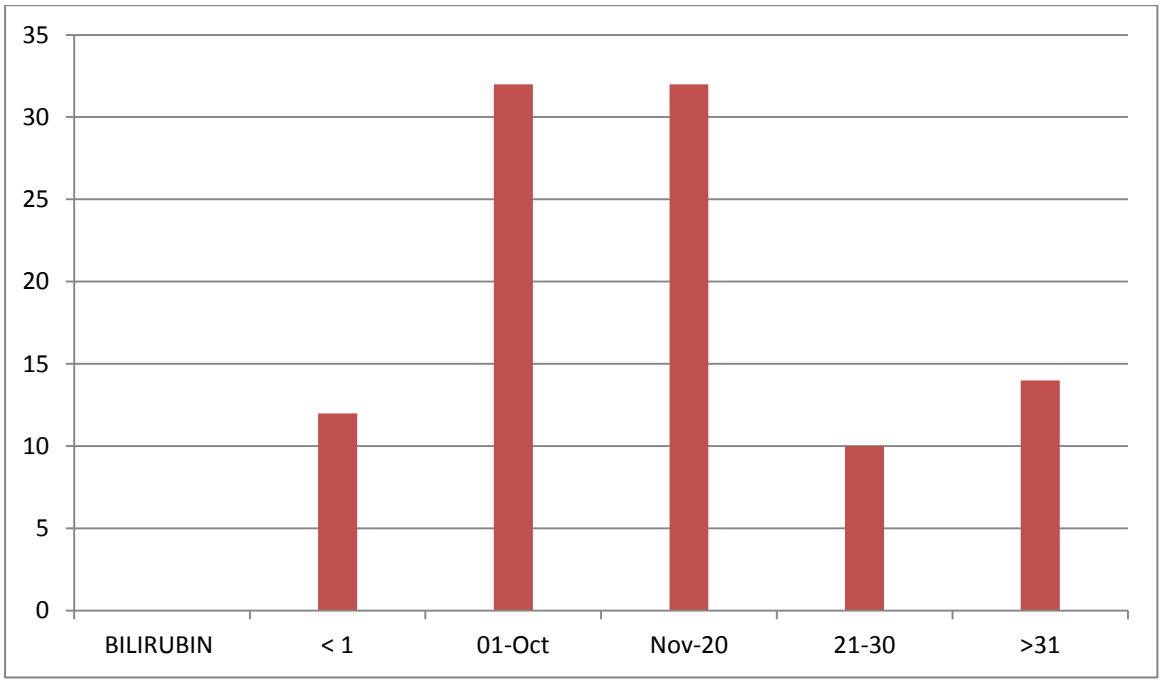
PATIENTS PRESENTING WITH LOSS OF WEIGHT / LOSS OF APPETITE

Loss of weight & Loss of appetite	FREQUENCY	PERCENTAGE (%)
Present	14	28
Absent	36	72



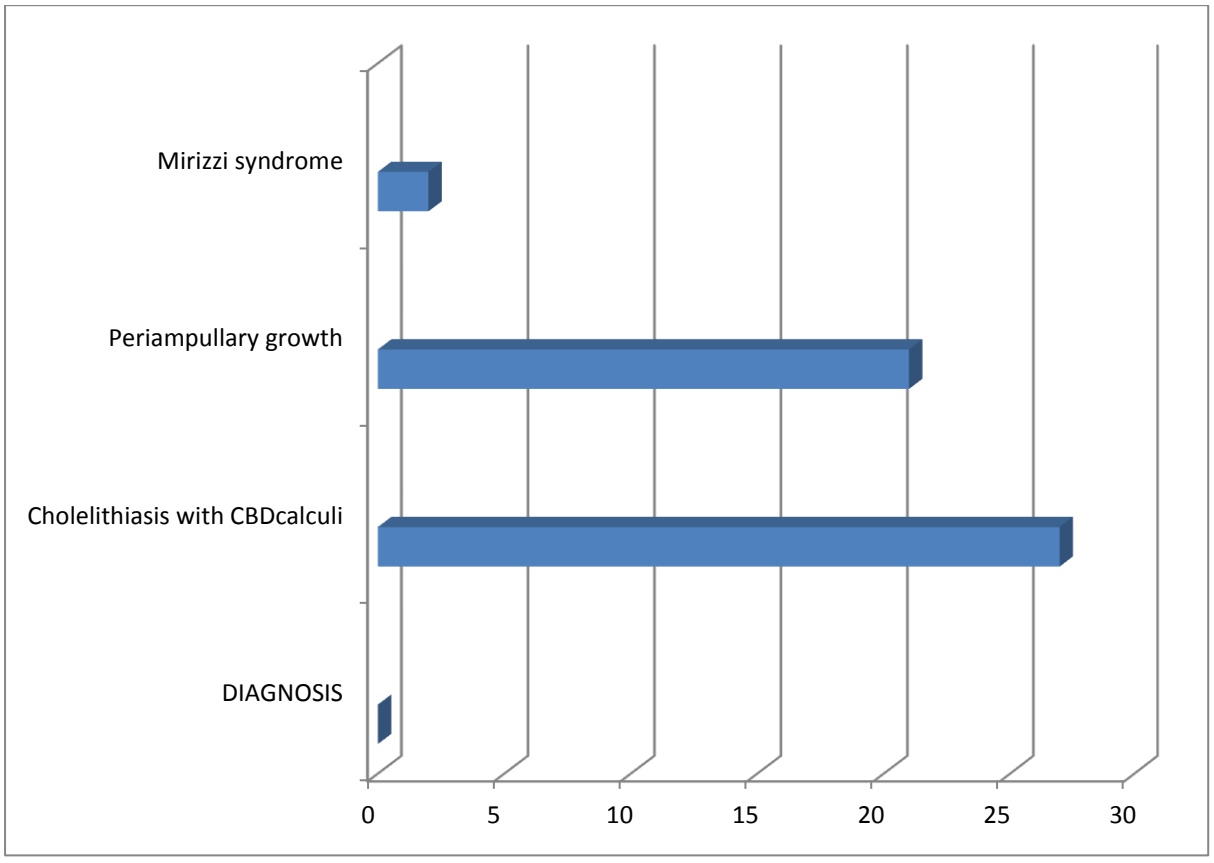
BILIRUBIN LEVELS OF PATIENTS

BILIRUBIN	FREQUENCY	PERCENTAGE
< 1	6	12
1- 10	16	32
11-20	16	32
21-30	5	10
>31	7	14



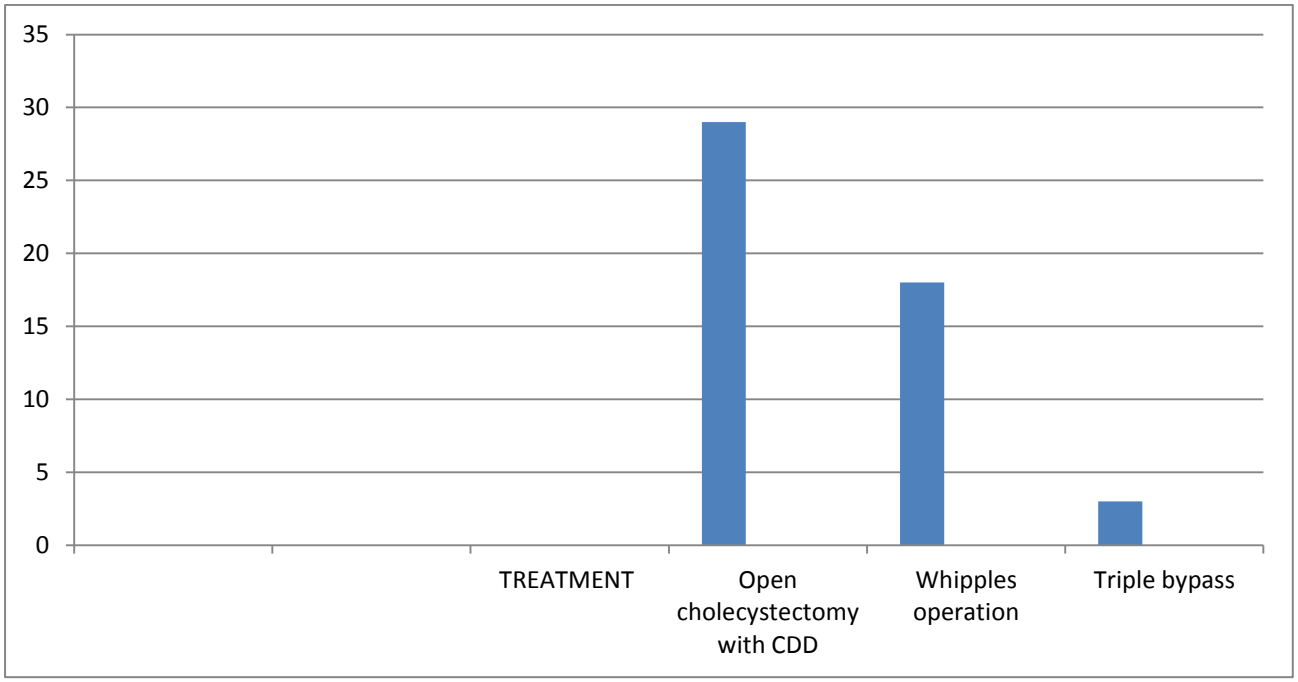
DIAGNOSIS

DIAGNOSIS	FREQUENCY	PERCENTAGE
Cholelithiasis with CBDcalculi	27	54
Periampullary growth	21	42
Mirizzi syndrome	2	4



TREATMENT

TREATMENT	FREQUENCY	TREATMENT
Open cholecystectomy with CDD	29	58
Whipples operation	18	36
Triple bypass	3	6



DISCUSSION

Obstructive jaundice is a frequent condition of biliary tract disorders and the evaluation and management of the jaundice patient is a common problem facing the General Surgeon. While diagnosing a case of surgical jaundice, a thorough history, a complete physical examination & biochemical tests are necessary. Once diagnosed, the surgeon should have good knowledge about the anatomy of the biliary tree, physiology of bile metabolism and pathophysiological change s occurring in the liver, secondary to obstruction, various causes of obstruction, different imaging facilities and different modalities of treatment.

In this study, analysis of the various causes of surgical jaundice & its presentation were done. Investigations were carried out and different ty pes of operative procedures were conducted. Total number of cases were 50. The results were compared with other similar studies done by various authors.

The common presentation in a benign condition was pain abdomen and flatulent dyspepsia, whereas in malignant condition it was jaundice, high coloured urine, pale stool & loss of appetite. Most malignant cases had Icterus and a palpable gall bladder when compared to benign condition.

There were significantly higher values of total bilirubin, direct Bilirubin and alkaline phosphatase in malignant conditions. It was found that obstructive jaundice secondary to common bile duct stones remains the commonest cause, obstructive jaundice secondary to malignancy was the second most common cause followed by benign stricture.

USG abdomen was carried out on all patients as a standard imaging technique for investigation on a patient presenting with jaundice. USG was successfully used as a cheapest non-invasive tool to know the cause and level of obstruction in nearly 93% of the patients (USG was unable to diagnose one benign CBD stricture which was diagnosed by ERCP). The limitation of this diagnostic test was its high operator dependence.

ERCP: Its value is its ability to remove stones, stenting and also taking tissue for HPE. ERCP is also one of the diagnostic tools used for surgical jaundice.

MRCP: It's a diagnostic test for imaging of biliary tree. Drawback of this imaging technique is its inability to remove calculi, stenting or biopsy tissue for HPE.

CT scan was also used in selected cases to confirm the diagnosis made on USG. Patient with obstructive jaundice due to CBD calculi underwent Cholecystectomy with CBD exploration with either T-tube dr ainage after

intraoperative cholangiogram showed normal flow of dye into duo denum with no residual calculi or choledochoduodenostomy.

Definitive procedure done for benign CBD stricture following Cholecystectomy was Hepatico-Jejunostomy with enteroenterostomy.

Obstructive jaundice due to malignanc y, 3underwent palliative procedure and 18 patients underwent definitive procedure (Whipple's procedure). The outcome of palliative procedures was good. Patients were free from jaundice.

SUMMARY

50 patients were diagnosed to have su rgical jaundice in the study period from july2013 to july 2014. Th e study was conducted at the institute of Surgery. A brief introduction and a hist orical review of biliary tract has been presented with a detailed discussion on the surgical anatomy, physiology, etiopathogenesis, clinical features, investigations and management of surgical jaundice. The findings of this study were compared with those available in literature. The results have been represented with tables and graphs for better understanding.

The findings of the study are as follows:

1. The occurrence of surgical jaundice was maximum in the 41-50 year age group.

2.clinical Icterus was present in 34 patients . Pain abdomen and flatulent dyspepsia were more in benign condition whereas jaundice, clay-coloured stools, high coloured urine with itching was more common in malignancy.

3. High values of serum Bilirubin & alkaline phosphatase are noted in malignancy.

4. USG was the cheapest non-invasive investigation used for diagnosis of surgical jaundice.

5. Most common cause of obstruction was CBD calculi, followed by malignancy, then by benign CBD stricture.

6. For CBD calculi, CBD exploration with cholecystectomy and drainage procedure was done by T tube or choledochoduodenostom

7. For malignancy operative curative pr ocedure was Whipple's surgery and

palliative procedure was triple byepass

8. Recently increasing reliance of ERCP and MRCP to image biliary tract has helped to diagnose the pathology earlier and hence early intervention can be initiated.

CONCLUSION

Common presentation of surgical jaundice is jaundice.

Palpable GB indicates the etiology to be malignant.

Common cause for surgical jaundice is CBD calculi.

USG remains the cheapest, safest and mo st reliable diagnostic tool in the management of surgical jaundice.

Open exploration of CBD under experienced hands is a good treatment modality in the management of obstructive jaundice.

In malignancies, early detection and staging and proper selection of the patient are more important to gain benefit from resection of tumour, whereas late presentation and those patients not suitable for resection had good improvement in quality of survival with palliative surgery.

Improving deranged LFT, correction of anaemia and hepatorenal problem improves the surgical results (morbidity and mortality).

BIBLIOGRAPHY

1. Scharsschmidt GF, Goldberg HI, Schmid R. Approach to the patient with cholestatic jaundice. N Engl J Med 1983; 308: 1515-1519.

2. Kim U. Kahnag, Joel J. Roslyn. Jaundice: Maingot's abdominal operations. Vol. I & II. 10th edition. Singapore: Mc Graw Hill; 2001. pg. 315-336, 1701-2031.

3. Morgenstern L. A history of choledochotom y. In: Berci G, Cushieri A. Bile ducts and Bile duct stones, WB Saunders 1997; 2: 3-8.

4. Wood MD. Presidential a ddress; Eponyms in biliary tract surgery. Am J Surg 1979; 138: 746-754.

5. Mulvihill SJ, Debas HT. History of surgery of gastrointestinal tract. In: Norton JA, Bollinger RR, et al (Eds). Surgery, Basic science and clinical evidence. Springer-Velrag 2001; 23: 399-412.

6. William PL, Warwick R, et al (Eds). Gray's Anatomy. 38 th ed. NY: Churchill Livingstone; 1995. p. 1394-96.

7. Adkins RB, Chapman WC, Reddy VS . Embryology, Anatomy and Surgical applications of the extrahepatic biliar y system. In: Skandalakis SE, Flament JB. Surgical Anatomy and Embryology. Surg Clin N Am Feb 2000; 80 (1): 363-374.

8. Inderbir Singh. Human Embryology, 7th ed. New Delhi: Current. p. 186-196.

9. Anne M. R. Agur, Ming J. Lee. Grant's Anatomy. 10th ed. Philadelphia: Lippincot William & Wilkins; 1999. p. 613-20.

10. Erlinger S. Bile secretion. In: Blumgart LH, et al. Su rgery of the liver and the biliary tract. 3rd ed. NY: Churchill Livingstone; 2000. 1 (5): 109-20.

11. Schwartz SI. Gallbladder and extrahepatic biliary system. In: Schwartz SI, Shires GT, Spencher FC et al (eds). Principles of Surgery. 7 th ed. Singapore: Mc Graw Hill; 1999. 2: 1437-1466.

12. Guyton C. Textbook of Medical Physiology, 10 th ed. Singapore: Harcourt; 2000. 749-52, 800-03.

13. Principles and practice of Medicine. Osler W. 5 th ed. New York: Appleton and

Co. 1992. p. 569.

14. Cotran RS, Kumar V, Robbins SL. Robbi ns Pathologic basis of disease. 6 th ed.

Philadelphia: WB Saunders; 1999.

15. Diliberto JP, Marks JW, Schoenfield LJ. Classification and pathogenesis of

gallstones. In: Braasch JW, Tompkins RK. Surgical disease of the biliary tract and

pancreas. Chicago IL; 1994.

16. Lordsmith of Marlow, Sheila Sherlock. Su rgery of the gallbladder and bile ducts.

2nd ed. London: Butterworth; 1981.

17. Saunders KD, Cates JA, Roslyn JJ. Pathogenesis of gallstones. In: Pitt HA. The

Surgical Clinics of North America: Bilia ry tract surgery, Philadelphia, PA: WB

Saunders 1990; 1197-1216.

18. Smith Al, Stewart L, Fine R, et al. Gallst ones disease. The clinical manifestations

of infectious stones. Arch Surg 1989; 124: 169-633.

19. Kaufman HS, Magnuson TH, Lillemoe KD et al. The role of bacteria in gallbladder and common duct stone formation. Ann Surg 1989; 209: 584-591.

20. Khuroo MS, Sarjar SA, Mahajan R. Hepatobiliary and pancreatic ascariasis in India. Lancet 1990; 335: 1503-6.

21. Choi BI, Park JH, Kim YI, et al. Peripheral cholangiocarcinoma and clonorchiasis: CT findings. Radiology 1998; 189: 149-153.

22. Howard ER. Surgery of liver dis ease in childhood. Oxford: Butterworth-Heinemann; 1991.

23. Howard ER. Biliary Atresia. In: Blum gart LH, Fong Y. Surgery of Liver & Biliary Tract. 3rd Ed. London: WB Saunders; 2000. p. 853-77.

24. Chijlwa K, Koga A. Surgical management and long-term follow up of patients with choledochol cyst. Am J Surg 1993; 165: 238-242.

25. Lennot JP, et al. Bile Duct Cysts in Adults. Ann of Surg 1998; 228(2):159-66.

26. Tondelli P, Schuppisier JP. Papillary ste nosis. In: Blumgart LH, Fong Y(eds)

Surgery of the liver and biliary tract. Edinburgh: Churchill Livingstone; 1998.

27. Kune GA, Sali A. Benign biliary strictur es. In: The practice of biliary surgery. 2 nd

ed. Oxford: Blackwell Scientific; 1981.

28. Blumgart LH, Kelley CJ, Benjamin IS. Benign bile duct stricture following

cholecystectomy: Critical factors in management. Br J Surg 1984; 71: 836.

29. Blumgart LH. Bile duct injuries/stricture. In: Fromm D (ed) Gastrointestinal

Surgery Vol. 2, London: Churchill Livingstone; 1984. p. 755.

30. Smith R. Obstructions of the bile ducts. Br J Surg 1979; 69: 317.

PROFORMA

CLINICAL STUDY AND MANAGEMENT OF OBSTRUCTIVE JAUNDICE

1. Name: Age : Sex :
2. Address: IP. No:
3. DOA: DOO: DOD :
4. Occupation:
5. Chief complaints:
6. History of Present Illness:

A. Pain. B. Flatulent Dyspepsia C.Jaundice D.itching

Duration:

Mode of onset:

Belching:

Heart Burn:

Clay-coloured urine

Nausea/ vomiting

Duration: Character:

Relation to food:

Relieving factors Relation with pain:

High coloured urine:

G. Haematemesis / Melena

H. Fever

Weight Loss:

Loss of appetite:

11. Per Abdominal Examination

Contour Skin over the abdomen

Abdominal movement: Respiratory: Peristaltic:

Pulsatile:

Umbilicus Visible mass:

Palpation

If Mass Present:

Number: Site: Size:

Shape: Surface: Extent:

Consistency: Margins: Tenderness:

Movement with respiration:

Plane of the swelling:

Free Fluid:

Percussion

Liver dullness

Shifting dullness

Percussion over the mass

Auscultation Per-rectal examination:

Bowel sounds

Bruit Per-vaginal examination:

Respiratory System Cardiovascular System

Central Nervous System

DIAGNOSIS:

TREATMENT

Procedure done:

Operative findings:

HPE Report:

LIST OF ABBREVIATIONS USED

A/G Albumin – Globulin ratio

Ca Carcinoma

CBD Common bile duct

CT Computed Tomography

ECBD Exploration of the CBD

dL Deciliter

EU Endoscopic Ultrasonography

ERCP Endoscopic retrograde Cholangiopancreatography

IU International Unit

lab Laboratory

mg Milligram

MRCP Magnetic resonance Cholangiopancreatography

PTC Percutaneous transhepatic cholangiography

PT Prothrombin time

SD Standard Deviation

SGOT Serum Glutamic Oxaloacetic Transaminase

USG Ultrasonography

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI-3

EC Reg No.ECR/270/Inst./TN/2013
Telephone No. 044 25305301
Fax : 044 25363970

CERTIFICATE OF APPROVAL

To
Dr.K.M.Balaji,
Postgraduate MS (General Surgery),
Institute of General Surgery,
Madras Medical College,
Chennai – 600 003.

Dr.K.M.Balaji,

The Institutional Ethics Committee has considered your request and approved your study titled **"Clinical study and management of obstructive jaundice"** No.23072014.

The following members of Ethics Committee were present in the meeting held on 01.07.2014 conducted at Madras Medical College, Chennai-3.

1.	Dr.C.Rajendran, M.D.,	: Chairperson
2.	Dr.R.Vimala, M.D., Dean, MMC, Ch-3	: Deputy Chairperson
3.	Prof.B.Kalaiselvi, M.D., Vice-Principal, MMC, Ch-3	: Member Secretary
4.	Prof.R.Nandhini, M.D., Inst.of Pharmacology, MMC	: Member
5.	Dr.G.Muralidharan, Director Incharge, Inst.of Surgery	: Member
6.	Prof.Md.Ali, M.D., D.M., Prof & HOD of MGE, MMC	: Member
7.	Prof.K.Ramadevi, Director i/c, Inst.of Biochemistry, MMC	: Member
8.	Prof.Saraswathy, M.D., Director, Pathology, MMC, Ch-3	: Member
9.	Prof.Tito, M.D., Director i/c, Inst.of Internal Medicine, MMC	: Member
10.	Thiru S.Rameshkumar, Administrative Officer	: Lay Person
11.	Thiru S.Govindasamy, B.A., B.L.,	: Lawyer
12.	Tmt.Arnold Saulina, M.A., MSW.,	: Social Scientist

We approve the proposal to be conducted in its presented form.

Sd/ Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

16/7/14

Member Secretary, Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003

ஆராய்ச்சி தகவல் தாள்

சென்னை இராஜீவ்காந்தி அரசு பொது மருத்துவனைக்கு வரும் நோயாளிகளின் மஞ்சள் காமாலை நோயின் தன்மை மற்றும் சிகிச்சை முறை பற்றிய ஆய்வு நடைபெறுகிறது.

நீங்களும் இந்த ஆராய்ச்சியில் பங்கேற்க நாங்கள் விரும்புகிறோம்.

இந்த ஆராய்ச்சியின் முடிவுகளை அல்லது கருத்துக்களை வெளியிடும்போதோ அல்லது ஆராய்ச்சியின்போதோ தங்களது பெயரையோ அல்லது அடையாளங்களையோ வெளியிட மாட்டோம் என்பதை தெரிவித்துக்கொள்கிறோம்.

இந்த ஆராய்ச்சியில் பங்கேற்பது தங்களுடைய விருப்பத்தின்பேரில்தான் இருக்கிறது. மேலும் நீங்கள் எந்த நேரமும் இந்த ஆராய்ச்சியிலிருந்து பின்வாங்கலாம் என்பதையும் தெரிவித்துக்கொள்கிறோம்.

இந்த ஆராய்ச்சியின் முடிவுகளை ஆராய்ச்சின் போது அல்லது ஆராய்ச்சின் முடிவின் போது தங்களுக்கு அறிவிக்கப்படும் என்பதையும் தெரிவித்துக்கொள்கிறோம்.

மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது. என்னுடைய சந்தேங்களை கேட்கவும், அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டது.

நான் இவ்வாய்வில் தன்னிச்சையாகத்தான் பங்கேற்கிறேன். எந்த காரணத்தினாலோ எந்த கட்டத்திலும் எந்த சட்ட சிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகி கொள்ளலாம் என்றும் அறிந்து கொண்டேன்.

ஆராய்ச்சியாளர் கையொப்பம் பங்கேற்பாளர் கையொப்பம்

நாள் :
இடம் :

ஆராய்ச்சி ஒப்புதல் படிவம்

ஆராய்ச்சி தலைப்பு

மஞ்சள் காமாலை நோயின் தன்மை மற்றும் சிகிச்சை முறை பற்றிய ஆய்வு

பெயர்	:	தேதி	:
வயது	:	உள் நோயாளி எண்	:
பால்	:	ஆராய்ச்சி சேர்க்கை எண்	:

இந்த ஆராய்ச்சியின் விவரங்களும் அதன் நோக்கமும் முழுமையாக எனக்கு தெளிவாக விளக்கப்பட்டது.

எனக்கு விளக்கப்பட்ட விஷயங்களை புரிந்துகொண்டு நான் எனது சம்மதத்தை தெரிவிக்கிறேன்.

இந்த ஆராய்ச்சிக்காக அல்ட்ராசவுண்ட், சி.டி., எம்.ஆர்.சி.பி., இ.ஆர்.சி.பி., ஸ்கேன் எடுக்கப்படும் என்பதை அறிந்துகொண்டேன்.

இந்த ஆராய்ச்சியில் பிறரின் நிர்பந்தமின்றி என் சொந்த விருப்பத்தின்பேரில் நான் பங்கு பெறுகின்றேன். இந்த ஆராய்ச்சியில் இருந்து நான் எந்நேரமும் பின் வாங்கலாம் என்பதையும் அதனால் எந்த பாதிப்பும் ஏற்படாது என்பதையும் நான் புரிந்துகொண்டேன்.

இந்த ஆராய்ச்சியின் விபரங்களைக் கொண்ட ஆராய்ச்சி தகவல்தாளைப் பெற்றுக்கொண்டேன்.

நான் என்னுடைய சுய நினைவுடனும் மற்றும் முழு சுதந்திரத்துடனும் இந்த மருத்துவ ஆராய்ச்சியில் என்னை சேர்த்துக்கொள்ள சம்மதிக்கிறேன்.

ஆராய்ச்சியாளர் கையொப்பம் **பங்கேற்பாளர் கையொப்பம்**

நாள் :
இடம் :

S.No	NAME	AGE	SEX	I.P NO:	Jaundice		Abdominal pain	Itching	Flatulent dyspepsia	Loss of appetite / Loss of weight	DIAGNOSIS	MANAGEMENT
					Clinical	Bilirubin mgs%						
1	Seetha	29	F	18116	-	1	+	-	+	-	Cholelithiasis with cbd calculi	Open cholecystectomy with cbd exploration
2	Raja kumari	35	F	71258	+	6.1	-	-	-	-	Cholelithiasis with cbd calculi	Open cholecystectomy with cbd exploration
3	Ranganathan	40	M	22889	+	7.4	+	-	-	-	Cholelithiasis with cbd calculi	Open cholecystectomy with cbd exploration
4	Mary	42	F	17535	+	10.5	-	-	-	-	Periampullary growth	whipples operation
5	Mukayan	43	M	20256	-	1.8	-	-	+	-	Cholelithiasis with cbd calculi	Open cholecystectomy with cbd exploration
6	Annammal	40	F	20354	+	12	-	-	-	-	Cholelithiasis with cbd calculi	Open cholecystectomy with cbd exploration
7	Subramaniyan	42	M	27872	+	16	+	+	-	+	Periampullary growth	whipples operation
8	Marimuthu	40	M	59030	+	9	-	-	-	-	Cholelithiasis with cbd calculi	Open cholecystectomy with cbd exploration
9	Rani	50	F	40789	-	0.9	-	-	+	-	Cholelithiasis with cbd calculi	Open cholecystectomy with cbd exploration

10	Govindan	50	M	25944	+	21	+	+	-	-	Periampullary growth	whipples operation
11	Moorthy	52	M	22458	-	0.7	-	-	-	-	Cholelithiasis with cbd calculi	Open cholecystectomy with cbd exploration
12	Kailasam	50	M	12741	+	15.9	+	+	-	+	Periampullary growth	Triple byepass
13	Raji	50	M	106480	+	19.2	+	+	-	+	Periampullary growth	whipples operation
14	Subramani	56	M	104081	+	20.6	+	+	-	+	Periampullary growth	whipples operation
15	Kala	50	F	33579	-	3.9	-	-	+	-	Cholelithiasis with cbd calculi	Open cholecystectomy with cbd exploration
16	Bakthavachalam	55	M	35182	-	0.9	-	-	+	-	Cholelithiasis with cbd calculi	Open cholecystectomy with cbd exploration
17	Chandrasekar	57	M	69308	+	23	+	+	-	+	Periampullary growth	whipples operation
18	Kajalaksmi	55	F	69518	-	0.8	-	-	+	-	Cholelithiasis with cbd calculi	Open cholecystectomy with cbd exploration
19	Jyothi	50	M	79795	+	14.8	-	-	-	-	Cholelithiasis with cbd stricture	open cholecystectomy with CDD
20	Annappan	50	M	3664	+	10.6	-	-	-	-	Cholelithiasis with cbd calculi	Open cholecystectomy with cbd exploration
21	Masthanama	50	F	15510	+	29.2	+	+	-	+	Periampullary growth	whipples operation
22	Padmavathi	57	F	20427	-	1.9	-	-	+	-	Cholelithiasis with cbd calculi	Open cholecystectomy with cbd exploration
23	Mani	59	M	32766	-	0.8	-	-	+	-	Cholelithiasis with cbd calculi	Open cholecystectomy with cbd exploration
24	Kuselan	52	M	27740	+	21.2	+	+	-	+	Periampullary growth	whipples operation
25	Pattabi	61	M	76152	+	17.2	-	+	-	-	Cholelithiasis with cbd calculi	Open cholecystectomy with cbd

												exploration
26	Sundaravalli	60	F	20270	+	19.2	-	-	-	-	Periampullary growth	whipples operation
27	maheshwari	39	F	112773	+	30.1	+	+	-	+	Mirizzi syndrome	open cholecystectomy with CDD
28	Kasi	65	M	67059	+	28.8	+	+	-	+	Periampullary growth	whipples operation
29	Subramani	62	M	74923	-	2.9	-	-	+	-	Cholelithiasis with cbd calculi	Open cholecystectomy with cbd exploration
30	Ponnammal	63	F	34571	+	17.3	+	+	-	-	Periampullary growth	Whipples operation
31	Sulochana	60	F	35456	+	10	-	-	-	-	Cholelithiasis with cbd calculi	Open cholecystectomy with cbd exploration
32	Raman	69	M	23283	-	4.9	-	-	-	-	Cholelithiasis with cbd calculi	Open cholecystectomy with cbd exploration
33	Ayyapan	60	M	74935	+	7.9	-	-	-	-	Cholelithiasis with cbd calculi	Open cholecystectomy with cbd exploration
34	Venkatesan	70	M	118807	+	9.6	-	-	-	-	Periampullary growth	Triple byepass
35	srinivasan	45	M	116400	+	32.0	+	+	-	+	Mirizzi syndrome	open cholecystectomy with CDD
36	Krishnan	72	M	76501	+	20.6	+	+	-	+	Periampullary growth	whipples operation
37	Govindasamy	62	M	111211	-	4.9	-	-	+	-	Cholelithiasis with cbd calculi	Open cholecystectomy with cbd exploration
38	Palani	76	M	78481	+	15.4	-	-	-	-	Periampullary growth	whipples operation
39	Chinnaiyan	57	M	10265	+	16.4	+	-	-	-	Periampullary growth	whipples operation
40	Ponni	35	F	11195	-	3.7	-	-	-	-	Cholelithiasis with cbd calculi	Open cholecystectomy with cbd exploration
41	Varalaksmi	35	F	45984	-	3.3	-	-	+	-	Cholelithiasis with cbd calculi	Open cholecystectomy with cbd exploration

42	Balu	49	M	88345	+	12.9	-	-	-	-	Cholelithiasis with cbd calculi	open cholecystectomy with CDD
43	Mariammal	37	F	11195	+	18.4	+	+	-	+	Periampullary growth	whipples operation
44	Subramani	65	M	30350	-	5.0	-	-	+	-	Cholelithiasis with cbd calculi	Open cholecystectomy with cbd exploration
45	Lakshmi	55	F	31175	+	14.9	-	-	-	-	Periampullary growth	triple byepass
46	Parveen	41	F	38073	+	19.0	+	+	-	-	Periampullary growth	whipples operation
47	Kanniammal	45	F	35421	-	4.7	-	-	+	-	Cholelithiasis with cbd calculi	Open cholecystectomy with cbd exploration
48	Mariappan	48	M	75638	+	20.7	+	+	-	+	Periampullary growth	whipples operation
49	Palanivel	54	M	112690	+	13.9	-	-	-	-	Periampullary growth	whipples operation
50	Srividya	47	F	33172	+	12.6	-	-	-	-	Cholelithiasis with cbd calculi	Open cholecystectomy with CDD

CLINICAL STUDY AND MANAGEMENT OF OBSTRUCTIVE JAUNDICE

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INTRODUCTION

Jaundice is a frequent manifestation of biliary tract disorders and the evaluation and management of obstructive jaundice is a common problem faced by the general surgeon.

Obstructive jaundice is strictly defined as a condition occurring due to a block in the pathway between the site of conjugation of bile in liver cells and the entry of bile into the duodenum through the ampulla. The block may be intrahepatic or extra hepatic in the bile duct.1.

Despite the technical advances, the operative modes of management of obstructive jaundice were associated with very high morbidity and mortality. Yet, during the last decade significant advances have been made in our understanding with regard to the pathogenesis, diagnosis, staging and the efficacy of management of obstructive jaundice.2 .

Obstructive jaundice of varied etiology is one of the cause s of admission to hospitals across North Karnataka. To diagnose the cause, site of obstruction and management of a case of surgical jaundice is indeed a challenging task for